

HIV Monoclonal Antibodies

Table 10: gp120

MAb ID	Location	WEAU	Sequence	Neutralizing	ImmunoGen	Species(Isootype)
195 M85	gp120(C1 30-51 LAI)	gp120(29-50)	ATEKLWVTVYYGVPV-WKEAFTTT	N	451 Env	murine(IgG1)
Donor: Fulvia di Marzo Veronese						
References: [di Marzo Veronese et al.(1992), Moore et al.(1994c), Moore et al.(1994d), Moore & Sodroski(1996), Ditzel et al.(1997)]						
NOTES:						
• M85: Immunoblot and RIP reactive for strains IIIB, 451, MN, RF, and RUTZ – binds deglycosylated gp120 [di Marzo Veronese et al.(1992)]						
• M85: C1 domain – mutation 40 Y/D impairs binding – the relative affinity for denatured/native gp120 is < .01, suggesting conformational component [Moore et al.(1994c)]						
• M85: Binding inhibited by MAb 4D4#85, enhanced by conformationally sensitive anti-V3 MAb 5G11, and some anti-18 MAbs [Moore & Sodroski(1996)]						
196 7E2/4	gp120(C1 31-50 LAI)	gp120(30-49)	TEKLWVTVYYGVPVW-KEATT	Env glycopro		murine(IgG)
Donor: S. Ranjbar, NIBSC, UK						
References: [Moore et al.(1994c)]						
NOTES:						
• 7E2/4: C1 domain – the relative affinity for denatured/native gp120 is .07, suggesting conformational component [Moore et al.(1994c)]						
• 7E2/4: UK Medical Research Council AIDS reagent: ARP3050						
197 M92	gp120(C1 31-50 LAI)	gp120(40-49)	GVPVWKEATT	N	451 Env	rat(IgG1)
Donor: Fulvia di Marzo Veronese						
References: [di Marzo Veronese et al.(1992), Moore et al.(1994c), Moore et al.(1994d)]						
NOTES:						
• M92: Immunoblot reactive, RIP negative, but precipitates deglycosylated gp120 – reacts with strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese et al.(1992)]						
• M92: The relative affinity for denatured/native gp120 is 1 [Moore et al.(1994c)]						

MAb ID	Location	WEAU	Sequence	Neutralizing	Immuno	Species(Isotype)
198 4D4#85	gp120(C1 31-50 LAI)	gp120(40-49)	GVPVWKEATT	Env		murine(IgG)
Donor:	S. Nigida and L. Arthur, NCI, Frederick, MD USA					
References:	[Moore et al.(1994c), Moore et al.(1994d), Moore & Sodroski(1996)]					
NOTES:						
• 4D4#85: 4D4#85: C1 domain – the relative affinity, denatured/native gp120 is 0.1 – mutation 45 W/S impairs binding [Moore et al.(1994c)]						
• 4D4#85: 4D4#85: Inhibits binding of C1 MAb M85, C1-C5 discontinuous epitope MAbs 181 and 212A, and CD4 binding induced MAbs 48d and 17b [Moore & Sodroski(1996)]						
199 M86	gp120(C1 42-61 LAI)	gp120(41-60)	VPVWKEATTTLFCAS-DAKAY	N	451 Env	murine(IgG ₁)
Donor:	Fulvia di Marzo Veronese					
References:	[di Marzo Veronese et al.(1992), Moore et al.(1994c)]					
NOTES:						
• M86: Immunoblot and RIP reactive for strains IIIB, 451, MN, RF, and RUTZ – binds deglycosylated gp120 [di Marzo Veronese et al.(1992)]						
• M86: C1 domain – the relative affinity for denatured/native gp120 is 1 [Moore et al.(1994c)]						
200 133/11	gp120(C1 64-78)	gp120(63-77)	EVHNWVWATHACVPTD	L	IIIB gp120	murine(IgG ₁)
References:	[Niedrig et al.(1992b)]					
NOTES:						
• 133/11: Region of overlap for reactive peptides is WATHA – weak neutralization of lab strains [Niedrig et al.(1992b)]						
201 133/237	gp120(C1 51-70 LAI)	gp120(60-69)	YDTEVHNWVA	L	IIIB gp120	murine(IgG ₁)
Donor:						
References:	[Niedrig et al.(1992b), Moore et al.(1994c), Moore et al.(1994d)]					
NOTES:						
• 133/237: Region of overlap for reactive peptides is WATHA – weak neutralization of lab strains [Niedrig et al.(1992b)]						
• 133/237: The relative affinity, denatured/native gp120 is 1.4 – mutation of position 69 W/L impairs binding [Moore et al.(1994c)]						

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
202 133/290	gp120(C1 61-70 LAI)	gp120(60-69)	YDTEVHNWVA	L	IIIIB gp120	murine(IgG ₁)
	References: [Niedrig et al.(1992b), Thali et al.(1993), Moore et al.(1994c), Moore et al.(1994d), Wyatt et al.(1995), Binley et al.(1997)]					
	NOTES:					
	<ul style="list-style-type: none"> • 133/290: Region of overlap for reactive peptides is WATHA – weak neutralization of lab strains [Niedrig et al.(1992b)] • 133/290: The relative affinity for denatured/native gp120 is 2.2 – mutation in position 69 W/L impairs binding [Moore et al.(1994c)] • 133/290: Used for antigen capture assay, either to bind gp120 to the ELISA plate, or to quantitate bound gp120 [Wyatt et al.(1995)] • 133/290: Reciprocal binding inhibition with the antibody 522-149, that binds to a discontinuous epitope – binding is enhanced by some C5 and C1 binding site antibodies [Moore & Sodroski(1996)] • 133/290: A high avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)] 					
203 D/3G5	gp120(C1 73-82 LAI)	gp120(72-81)	ACVPTIDPNPQ	N	Baculovirus-expressed rgp120 LAI	murine(IgG ₁)
	References: [Bristow et al.(1994)]					
	NOTES:					
	<ul style="list-style-type: none"> • D/3G5: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)] 					
204 D/6A11	gp120(C1 73-82 LAI)	gp120(72-81)	ACVPTIDPNPQ	N	Baculovirus-expressed rgp120 LAI	murine(unk)
	References: [Bristow et al.(1994)]					
	NOTES:					
	<ul style="list-style-type: none"> • D/6A11: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)] 					
205 D/5E12	gp120(C1 73-92 LAI)	gp120(72-91)	ACVPTDPNPQEVE- VLVNVTEN	N	Baculovirus-expressed rgp120 LAI	murine(unk)
	References: [Bristow et al.(1994)]					
	NOTES:					
	<ul style="list-style-type: none"> • D/5E12: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)] 					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
206 4A7C6	gp120(C1 81-90 LAI)	gp120(80-89)	PQEVVVLVNVNT	Env glycopro	murine(IgG)	
Donor: R. Tedder						
References: [Thianniart et al.(1989), Thali et al.(1993), Moore & Ho(1993), Moore et al.(1994c), Moore et al.(1994d), Moore & Sodroski(1996)]						
NOTES:						
	<ul style="list-style-type: none"> • 4A7C6: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] • 4A7C6: The relative affinity for denatured/native gp120 is 7.9 – mutation 88 N/P impairs binding [Moore et al.(1994c)] • 4A7C6: C1 region epitope (88 N/P substitutions abrogates binding), but substitutions 380 G/F and 420 I/R also impaired binding [Moore et al.(1994d)] • 4A7C6: Reciprocal binding inhibition with the antibody 133/192 – enhanced by anti-C5 antibodies, and C1 antibody 135/9 [Moore & Sodroski(1996)] • 4A7C6: UK Medical Research Council AIDS reagent: ARP 360 					
207 B242	gp120(C1 83-92 LAI)	gp120(82-91)	EVVVLVNVNTEN	N	Baculovirus-expressed mis-folded rgp160 IIIB:NL43, MicroGenSys	murine(IgG1)
References: [Bristow et al.(1994)]						
NOTES:	<ul style="list-style-type: none"> • B242: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)] 					
208 1D10	gp120(C1 81-100 LAI)	gp120(80-99)	PQEVVVLVNVNTEN- FDMWKNDM	L	IIIB-rgp120	rat(unR)
References: [Dowbenko et al.(1988), Berman et al.(1991), Nakamura et al.(1992), Moore et al.(1994c)]						
NOTES:						
	<ul style="list-style-type: none"> • 1D10: Cross-blocks 5B3 in IIIB-rgp160 ELISA – type specific in rgp120 ELISA binding [Nakamura et al.(1992)] • 1D10: The relative affinity for denatured/native gp120 is 13 – mutation 88 N/P impairs binding [Moore et al.(1994c)] 					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
209 133/192	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM	L	IIIIB gp120	murine(IgG1)
Donor: Matthias Niedrig						
References: [Niedrig et al.(1992b), Moore et al.(1993b), Moore et al.(1994c), Moore & Sodroski(1996), Trkola et al.(1996a), Binley et al.(1997)]						
NOTES:						
	<ul style="list-style-type: none"> • 133/192: Epitope seems complex, binds multiple peptides – weak neutralization of lab strain [Niedrig et al.(1992b)] • 133/192: The relative affinity for denatured/native gp120 is 1.8 [Moore et al.(1994c)] • 133/192: C1 region – substitutions 76P/Y, 113 D/A or R, 117 K/W, 420 I/R, 427 W/S impair binding, other substitutions enhanced binding [Moore et al.(1994d)] • 133/192: Reciprocal binding inhibition with the antibody 4A7C6 – enhanced by some anti-C5 and-C1 antibodies [Moore & Sodroski(1996)] • 133/192: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)] • 133/192: A low avidity C1 antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)] 					
210 C6	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM	mis-folded LAI rgp160	murine(IgG1)	
	References: [Abacioglu et al.(1994), Moore et al.(1994c), Pincus et al.(1996)]					
NOTES:						
	<ul style="list-style-type: none"> • C6: Also called Ch6? • C6: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [Abacioglu et al.(1994)] • C6: The relative affinity for denatured/native gp120 is 0.9 [Moore et al.(1994c)] • C6: There is FNM/FDM polymorphism in LAI-based peptides – N is essential (J. P. Moore, per. comm.) • C6: Called Ch6 – binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing [Pincus et al.(1996)] • C6: NIH AIDS Research and Reference Reagent Program: 810 					
211 B2	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM	mis-folded LAI rgp160	murine(IgG2b)	
	References: [Thali et al.(1993), Abacioglu et al.(1994), Moore et al.(1994c), Moore et al.(1994d), Binley et al.(1997)]					
NOTES:						
	<ul style="list-style-type: none"> • B2: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [Abacioglu et al.(1994)] • B2: The relative affinity for denatured/native gp120 is 1.4 [Moore et al.(1994c)] • B2: There is FNM/FDM polymorphism in LAI-based peptides, and N is essential (J. P. Moore, per. comm.) • B2: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)] 					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species (Isotype)
212 GV4D3	gp120(92-100 IIIB)	gp120(91-99)	NFNMWKNDM	gp120 complexed with MAb M77	murine(unk)	
References:	[Denisova et al.(1996)]					
NOTES:						
• GV4D3: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes – MAbs GV4H4 and GV5F9 are homologous to GV4D3 and were generated in the same experiment [Denisova et al.(1996)]						
213 D/4B5	gp120(C1 93-101 LAI)	gp120(92-100)	FNMWKNDM	N	Baculovirus-expressed rgp120 LAI	murine(unk)
References:	[Bristow et al.(1994)]					
NOTES:						
• D/4B5: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)]						
214 D/6B2	gp120(C1 93-101 LAI)	gp120(92-100)	FNMWKNDM	N	Baculovirus-expressed rgp120 LAI	murine(IgG1)
References:	[Bristow et al.(1994)]					
NOTES:						
• D/6B2: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)]						
215 D/5A11	gp120(C1 93-101 LAI)	gp120(92-100)	FNMWKNDM	N	Baculovirus-expressed rgp120 LAI	murine(unk)
References:	[Bristow et al.(1994)]					
NOTES:						
• D/5A11: C1 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)]						
216 B9	gp120(C1 93-96 LAI)	gp120(92-95)	FNMW		mis-folded LAI rgp160	murine(IgG1)
References:	[Abacioglu et al.(1994)]					
NOTES:						
• B9: C1 region – epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]						
217 B10	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDWKNDM		mis-folded LAI rgp160	murine(IgG1)
References:	[Abacioglu et al.(1994), Moore et al.(1994c)]					
NOTES:						
• B10: C1 region – epitope boundaries mapped by peptide scanning, FN MW core [Abacioglu et al.(1994)]						
• B10: The relative affinity for denatured/native gp120 is 0.4 [Moore et al.(1994c)]						
• B10: There is FN M/FDM polymorphism in LAI-based peptides, and N is essential (J. P. Moore, per. comm.)						

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
218 L5.1	gp120(C1 89-103 IIIB)	gp120(78-92)	PNPQEVVVLVNVTFNF	vaccinia gp160	murine(IgG)	
	References: [Akerblom et al.(1990)]					
219 B27	gp120(C1 94-97 BH10)	gp120(92-95)	FNMW	N	Baculovirus-expressed mis-folded rgp160 IIIB: NL43, MicroGenSys	murine(IgG ₁)
	References: [Abacioglu et al.(1994), Bristow et al.(1994)]					
	NOTES:					
	• B27: C1 region – epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					
	• B27: MAbs generated in the context of a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)]					
220 B35	gp120(C1 94-99 BH10)	gp120(92-97)	FNMWKN	mis-folded LAI rgp160	murine(IgG)	
	References: [Abacioglu et al.(1994)]					
	NOTES:					
	• B35: C1 region – epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					
221 489.1(961)	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM	Env	murine(IgG)	
	Donor: C. Bruck, SKB, Belgium					
	References: [Moore et al.(1994c)]					
	NOTES:					
	• 489.1(961): 489.1(961): The relative affinity for denatured/native gp120 is 1 [Moore et al.(1994c)]					
	• 489.1(961): 489.1(961): NIH AIDS Research and Reference Reagent Program: 961					
222 T1.1	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM	vaccinia gp160	murine(IgG)	
	References: [Akerblom et al.(1990), Brolden et al.(1990), Moore et al.(1994c)]					
	NOTES:					
	• T1.1: Also reacted in solid phase with gp120(234-248) NGTGPCTNVSTQCT [Akerblom et al.(1990)]					
	• T1.1: No ADCC activity – reactive peptide: NVTENFNMWKNDMVEQ, IIIB [Brolden et al.(1990)]					
	• T1.1: C1 region – the relative affinity for denatured/native gp120 is 1 [Moore et al.(1994c)]					
223 T7.1	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM	Env	murine(IgG)	
	References: [Akerblom et al.(1990), Bolmstedt et al.(1990), Moore et al.(1994c), Moore et al.(1994d)]					
	NOTES:					
	• T7.1: The relative affinity of denatured/native gp120 is 4.0 [Moore et al.(1994c)]					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
224 T9	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM	Env		murine(IgG)
Donor: Lennart Åkerblom, Britta Wahren and Jorma Hinkula						
References: [Åkerblom et al.(1990), Bolmstedt et al.(1990), Moore et al.(1994c), Moore et al.(1994d), Binley et al.(1997)]						
NOTES:						
<ul style="list-style-type: none"> • T9: The relative affinity of denatured/native gp120 is 7.9 [Moore et al.(1994c)] • T9: C1 region – 45 W/S, 88 N/P, 256 S/Y, 262 N/T, 475 M/S, 485 I/S, and 491 I/F enhanced binding, no substitution tested significantly inhibited [Moore et al.(1994d)] • T9: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)] 						
225 5B3	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM	N	IIIB-rspg160	murine(IgG)
References: [Berman et al.(1991), Nakamura et al.(1992), Beretta & Dagleish(1994), Moore et al.(1994c)]						
NOTES:						
<ul style="list-style-type: none"> • 5B3: Blocks gp120 -CD4 binding [Berman et al.(1991)] • 5B3: Cross-blocks 1D10 in competitive IIIB-rspg160 ELISA – no neutralization – blocks IIIB-gp120 sCD4 binding – localized binding to residues 72-106 [Nakamura et al.(1992)] • 5B3: The relative affinity of denatured/native gp120 is 8.3 [Moore et al.(1994c)] 						
226 MF49.1	gp120(C1 91-100 LAI)	gp120(90-99)	ENFDMWKNDM	Env		murine(IgG)
References: [Thianni et al.(1989), Moore et al.(1994c)]						
NOTES:						
<ul style="list-style-type: none"> • MF49.1: The relative affinity of denatured/native gp120 is 3.8 [Moore et al.(1994c)] 						
227 B20	gp120(C1 101-110 LAI)	gp120(100-109)	VEQMHEDIIS		mis-folded LAI rgp160	murine(IgG _{2a})
References: [Abacioglu et al.(1994), Moore et al.(1994c)]						
NOTES:						
<ul style="list-style-type: none"> • B20: C1 region – epitope boundaries mapped by peptide scanning – HEDII core [Abacioglu et al.(1994)] • B20: The relative affinity for denatured/native gp120 is 1 [Moore et al.(1994c)] 						

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
228 B18	gp120(C1 101-110 LAI)	gp120(100-109)	VEQVMHEDDIS	mis-folded LAI rgp160	murine(IgG _{2a})	
	References: [Abacioglu et al.(1994), Moore et al.(1994c)]					
NOTES:						
	• B18: C1 region – epitope boundaries mapped by peptide scanning, HEDII core [Abacioglu et al.(1994)]					
	• B18: The relative affinity for denatured/native gp120 is 1 [Moore et al.(1994c)]					
229 MF39.1	gp120(C1 101-110 LAI)	gp120(100-109)	VEQVMHEDDIS	Env	murine(IgG)	
	References: [Thiriar et al.(1989), Cook et al.(1994), Moore et al.(1994c)]					
NOTES:						
	• MF39.1: Called 39.1, and is probably the same as MF39.1 – MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)]					
	• MF39.1: The relative affinity of denatured/native gp120 is 30 [Moore et al.(1994c)]					
230 T2.1	gp120(C1 101-120 LAI)	gp120(100-119)	VEQVMHEDDIS-LWDQSLKPCV	Env	murine(IgG)	
	Donor: Lemnart Akerblom, Britta Wahren and Jorma Hinkula					
	References: [Akerblom et al.(1990), Bolmstedt et al.(1990), Moore et al.(1994c), Moore et al.(1994d)]					
NOTES:						
	• T2.1: The relative affinity for denatured/native gp120 is .27 – mutations 113 D/R, 106 E/A, and 117 D/A impair binding [Moore et al.(1994c)]					
231 11/65	gp120(311-321 HXB10)	gp120(101-120)	EQMHEDEIS-LWDQSLKPCVK	rgp120 BH10	rat(IgG _{2b})	
	References: [McKeating et al.(1992a)]					
NOTES:						
	• 11/65: Binds only soluble gp120, not virion bound – used to quantitate gp120 shedding – (numbering is incorrect in original?)					
	• 11/65: [McKeating et al.(1992a)]					
	• 11/65: UK Medical Research Council AIDS reagent: ARP3076					
232 6D8	gp120(C1 101-120 LAI)	gp120(100-119)	VEQVMHEDDIS-LWDQSLKPCV	IIIb-rgp120	rat(unk)	
	References: [Dowbenko et al.(1988), Nakamura et al.(1992), Moore et al.(1994c)]					
NOTES:						
	• 6D8: Highly cross reactive with multiple stains by rgp120 ELISA [Nakamura et al.(1992)]					
	• 6D8: The relative affinity for denatured/native gp120 is 15 – mutations 113 D/R and 113 D/A impair binding [Moore et al.(1994c)]					

MAb ID	Location	WEAU	Sequence	Neut	Immunogen	Species(Isotype)
233 M96	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDEIISLWD-QSLKPCV	N	451 Env	rat(IgG _{2a})
	Donor: Fulvia di Marzo Veronese					
	References: [di Marzo Veronese et al.(1992), Moore et al.(1994c), Moore et al.(1994d)]					
	NOTES:					
	• M96: Immunoblot reactive for strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese et al.(1992)]					
	• M96: C1 region – the relative affinity for denatured/native gp120 is 6 [Moore et al.(1994c)]					
234 37.1.1(ARP 327)	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDEIISLWD-QSLKPCV		Env glycopro	murine(IgG)
	Donor: Claudine Bruck					
	References: [Thiriant et al.(1989), Moore & Ho(1993), Moore et al.(1994c)]					
	NOTES:					
	• 37.1.1(ARP 327): 37.1.1: Called 37.1 – bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]					
	• 37.1.1(ARP 327): 37.1.1: The relative affinity for denatured/native gp120 is 8.6 – mutations 113 D/R (not D/A) and 117 K/W impair binding [Moore et al.(1994c)]					
	• 37.1.1(ARP 327): 37.1.1: UK Medical Research Council AIDS reagent: ARP327					
235 187.2.1	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDEIISLWD-QSLKPCV		Env glycopro	murine(IgG)
	Donor: Claudine Bruck and Clothilde Thiriant					
	References: [Thiriant et al.(1989), Moore & Ho(1993), Cook et al.(1994), Moore et al.(1994c), Moore et al.(1994d)]					
	NOTES:					
	• 187.2.1: Called 187.1, and is probably the same as 187.2.1 – bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]					
	• 187.2.1: Called 187.1, and is probably the same as 187.2.1 – MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)]					
	• 187.2.1: The relative affinity for denatured/native gp120 is 7 – mutations 113 D/A (not D/R) and 117 K/W impair binding [Moore et al.(1994c)]					
	• 187.2.1: UK Medical Research Council AIDS reagent: ARP332					
236 MF58.1	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDEIISLWD-QSLKPCV		Env	murine(IgG)
	References: [Thiriant et al.(1989), Moore et al.(1994c)]					

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237 MF77.1	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDISLWDQSLKPCV	Env		murine(IgG)
	References: [Thiriault et al.(1989), Moore et al.(1994c)]					
	NOTES:					
	• MF77.1: The relative affinity for denatured/native gp120 is 11 [Moore et al.(1994c)]					
238 MF119.1	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDISLWDQSLKPCV	Env		murine(IgG)
	References: [Thiriault et al.(1989), Moore et al.(1994c)]					
	NOTES:					
	• MF119.1: The relative affinity for denatured/native gp120 is 30 – mutations 113 D/A, 113 D/R, and 117 K/W impair binding [Moore et al.(1994c)]					
239 MF4.1	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDISLWDQSLKPCV	Env		murine(IgG)
	References: [Thiriault et al.(1989), Moore et al.(1994c)]					
	NOTES:					
	• MF4.1: The relative affinity for denatured/native gp120 is 8 [Moore et al.(1994c)]					
240 MF53.1	gp120(C1 101-120 LAI)	gp120(100-119)	VEQMHEDISLWDQSLKPCV	Env		murine(IgG)
	References: [Thiriault et al.(1989), Moore et al.(1994c)]					
	NOTES:					
	• MF53.1: The relative affinity for denatured/native gp120 is 10 [Moore et al.(1994c)]					
241 GV1A8	gp120(105-113 IIIB)	gp120(104-112)	HEDDISLWD	gp120 complexed with mAb M77		murine(unk)
	References: [Denisova et al.(1996)]					
	NOTES:					
	• GV1A8: When anti-V3 MAbs M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes – MAbs GV7A4 and GV5H5 are homologous to GV1A8 and were generated in the same experiment [Denisova et al.(1996)]					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
242 135/9	gp120(C1 111-120 LAI)	gp120(110-119)	LWDQSLKPCV	L	III B gp120	murine(IgG ₁)
Donor: Matthias Niedrig						
References: [Niedrig et al.(1992b), Moore et al.(1994c), Moore et al.(1994d), Moore & Sodroski(1996), Trkola et al.(1996a), Binley et al.(1997)]						
NOTES:						
	• 135/9: Defines the epitope as gp120(114-123) MHEDIISLWD (core LWD?) – weak neutralization of lab strain [Niedrig et al.(1992b)]					
	• 135/9: The relative affinity for denatured/native gp120 is 15 – mutation 113 D/R impairs binding to native and denatured, 113 D/A only to denatured [Moore et al.(1994c)]					
	• 135/9: Substitutions 106 E/A, 113 D/A or R, and 117 K/W impair binding, some substitutions enhance binding [Moore et al.(1994d)]					
	• 135/9: Binding is enhanced by some anti-C1 and anti-C5 antibodies – enhances binding of some anti-V3, anti-C4 and anti-V2 MAbs – 135/9 binds to predicted alpha-helix in C1 [Moore & Sodroski(1996)]					
	• 135/9: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola et al.(1996a)]					
	• 135/9: A high avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)]					
243 MF46.1	gp120(C1 101-120 LAI)	gp120(110-119)	LWDQSLKPCV	Env		murine(IgG)
References: [Thiriart et al.(1989), Moore et al.(1994c)]						
NOTES:						
	• MF46.1: The relative affinity for denatured/native gp120 is 8.5 [Moore et al.(1994c)]					
244 C4	gp120(C1 101-120 LAI)	gp120(110-119)	LWDQSLKPCV		mis-folded LAI rgp160	murine(IgG ₁)
Donor: George Lewis						
References: [Abacioglu et al.(1994), Moore & Ho(1993), Moore et al.(1994c)]						
NOTES:						
	• C4: Bound preferentially to denatured III B gp120 [Moore & Ho(1993)]					
	• C4: C1 region – epitope boundaries mapped by peptide scanning, BH10 core IIISLW [Abacioglu et al.(1994)]					
	• C4: The relative affinity for denatured/native gp120 is 10 [Moore et al.(1994c)]					
245 11	gp120(C1 101-120 LAI)	gp120(110-119)	LWDQSLKPCV	Env		murine(IgG)
References: [Thiriart et al.(1989), Moore et al.(1994c)]						
NOTES:						
	• 11: The relative affinity for denatured/native gp120 is 7.8 – mutation 113 D/R impairs binding [Moore et al.(1994c)]					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
246 12G10	gp120(C1 101-120 LAI)	gp120(110-119)	LWDQSLKPCCV	Env		murine(IgG)
	References: [Thiriart et al.(1989), Moore et al.(1994c)]					
NOTES:						
	• 12G10: The relative affinity for denatured/native gp120 is 17 – mutation 117 K/W impairs binding [Moore et al.(1994c)]					
247 7C10	gp120(C1 101-120 LAI)	gp120(110-119)	LWDQSLKPCCV	Env		murine(IgG)
	References: [Thiriart et al.(1989), Moore et al.(1994c)]					
NOTES:						
	• 7C10: The relative affinity for denatured/native gp120 is 5.8 – mutation 117 K/W impairs binding [Moore et al.(1994c)]					
248 W1	gp120(C1 102-121 LAI)	gp120(101-120)	EQMHEDIISLWDQSL _{L-} KPCVK	Env		murine(IgG)
	Donor: D. Weiner, U. Penn.					
	References: [Moore et al.(1994c)]					
NOTES:						
	• W1: The relative affinity for denatured/native gp120 is 6 – mutations 113 D/A, 113 D/R, and 117 K/W impair binding [Moore et al.(1994c)]					
249 T11	(C1 102-125)	gp120(101-124)	EQMHEDIISLWDQSL ₋ KPCVKLTPL	rec gp140		murine(unk)
	Donor: R. Doms, Univ. of Pennsylvania					
	References: [Earl et al.(1994), Jagodzinski et al.(1996)]					
NOTES:						
	• T11: Generated during a study of the influence of the oligomeric structure of Env in determining the repertoire of the Ab response – an oligomer with no gp120/gp41 cleavage site was used as the immunogen [Earl et al.(1994)]					
	• T11: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – deletion of the V3 loop from gp120 results in more potent T11 inhibition by CRDS [Jagodzinski et al.(1996)]					
250 B33	gp120(V2 123-142 LAI)	gp120(122-146)	TPLCVSLKCTDLGNA- TNTNS	N	Baculovirus-expressed mis-folded rgp160 IIIb:NL ₄₃ , MicroGenSys	murine(IgG _{2bκ})
	Donor: Daniels					
	References: [Abacioglu et al.(1994), Bristow et al.(1994)]					
NOTES:						
	• B33: There are two MAbs in the literature named B33. See also gp41, LAI 123-142 [Abacioglu et al.(1994)]					
	• B33: MAbs generated in the context of a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)]					
	• B33: UK Medical Research Council AIDS reagent: ARP304, gp160/41 binding					

MAb ID	Location	WEAU	Sequence	Neutralizing	ImmunoGen	Species(IsoType)
251 6D5	gp120(V2 122-141 LAI)	gp120(121-145)	LTPPLCVSLIKCT-DLKNDTNTN	Env		murine(IgG)
Donor: S. Nigida and L. Arthur, NCI, Frederick, MD USA References: [Moore et al.(1994c), Moore et al.(1994d)] NOTES:						
	• 6D5: The relative affinity for denatured/native gp120 is 15 – mutations Δ119-205 and 125 L/G impair binding [Moore et al.(1994c)]					
252 C108G	gp120(V2 162-169 HXB2)	gp120(166-173)	STSTRGKV	L	IIIB infection	chimpanzee(IgG _{1κ})
	References: [Warrier et al.(1994), Wu et al.(1995), Warrier et al.(1995), Warrier et al.(1996), Ugolini et al.(1997)] NOTES:					
	• C108G: High affinity, potent neutralization of HIV-1 IIIB – binding not affected by reduction of disulfide bonds – binding disrupted by removal of N-linked glycans – peptide binding lower affinity than glycosylated Env [Warrier et al.(1994)]					
	• C108G: Strain specificity: LAI, Bal, HXB2 – conformational character – glycosylation site at 160 critical – mutation of conserved glycosylation site at 156 increased epitope exposure [Wu et al.(1995)]					
	• C108G: Characterization of MAb variable region [Warrier et al.(1995)]					
	• C108G: Synergistic neutralization of HIV-1 when combined with anti-V3 MAbs 0.5β and C311E, or anti-CD4BS MAbs, 1125H and 5145A – neutralization further enhanced by presence of both 1125H and 0.5β [Warrier et al.(1996)]					
	• C108G: Viral binding inhibition by C108G was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]					
253 10/76b	gp120(V2 162-171 BH10)	gp120(166-174)	STSTRGKVQ	L (HXB10)	BH10 rgp120	rat(IgG _{2a})
	References: [McKeating et al.(1993b), McKeating et al.(1993a), Shotton et al.(1995), Wu et al.(1995), McKeating et al.(1996)] NOTES:					
	• 10/76b: R to L substitution abrogated binding – human sera recognize epitope [McKeating et al.(1993b)]					
	• 10/76b: Cross-competes with MAbs 10/76b and 11/4b – HXB2 neutralization escape mutant has the substitution I/T at residue 165 [Shotton et al.(1995)]					
	• 10/76b: Included in cross-competition and neutralization studies [Shotton et al.(1995)]					
	• 10/76b: HX10 strain specificity – binds native, deglycosylated, or dentured gp120 [Wu et al.(1995)]					
	• 10/76b: Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)]					
	• 10/76b: UK Medical Research Council AIDS reagent: ARP3077					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
254 11/4c	gp120(V2 162-171)	gp120(166-174)	STSIRGKVQ	L (HXB2)	BH10 gp120	rat(IgG _{2a})
	References: [McKeating et al.(1993b), Wu et al.(1995), Shotton et al.(1995)]					
NOTES:						
	<ul style="list-style-type: none"> • 11/4c: R to L substitution abrogated binding – human sera recognize epitope [McKeating et al.(1993b)] • 11/4c: HX10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu et al.(1995)] • 11/4c: Cross-competes with MAbs 10/76b and 11/4b – HXB2 neutralization escape mutant has the substitution I/T at residue 165 [Shotton et al.(1995)] • 11/4c: UK Medical Research Council AIDS reagent: ARP3035 					
255 11/4le	gp120(V2 162-171)	gp120(166-174)	STSIRGKVQ	L (HXB10)	gp120 LAI:BH10	rat(IgG ₁)
	References: [McKeating et al.(1993b), Shotton et al.(1995), Wu et al.(1995)]					
NOTES:						
	<ul style="list-style-type: none"> • 11/4le: R to L abrogated binding – human sera recognize the epitope [McKeating et al.(1993b)] • 11/4le: Included in cross-competition and neutralization studies [Shotton et al.(1995)] • 11/4le: HX10 strain specificity – binds native and deglycosylated gp120 [Wu et al.(1995)] 					
256 11/4b	gp120(V2 162-171)	gp120(166-174)	STSIRGKVQ	L (HXB10)	gp120 LAI:BH10	rat(IgG _{2a})
	References: [McKeating et al.(1993b), Shotton et al.(1995), Wu et al.(1995), Moore & Sodroski(1996)]					
NOTES:						
	<ul style="list-style-type: none"> • 11/4b: A change from R to L abrogated binding – human sera recognize epitope [McKeating et al.(1993b)] • 11/4b: Cross-competes with MAbs 10/76b and 11/4c – HXB2 neutralization escape mutant has the substitution I/T at residue 165 [Shotton et al.(1995)] • 11/4b: HX10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu et al.(1995)] • 11/4b: Linear V2 epitope – reciprocal binding enhancement of anti-V2 discontinuous epitope antibodies (in contrast to BAT085) and CD4-inducible antibody 48d. Reciprocal inhibits BAT085 binding – inhibits CRA-3 binding CRA-3 does not inhibit 11/4b [Moore & Sodroski(1996)] 					
257 RSD-33	gp120(V2 162-171 BH10)	gp120(166-174)	STSIRGKVQ		BH10 gp120	(unk)
	Donor: R. Daniels (NIMR, UK)					
	References: [Moore et al.(1993a)]					
NOTES:						
	<ul style="list-style-type: none"> • 6C4/S: UK Medical Research Council AIDS reagent: ARP3049 					
258 6C4/S	gp120(V2 162-170 BH10)	gp120(166-173)	STSIRGKV		BH10 gp120	(unk)
	Donor: S. Ranjbar (NIBSC, UK)					
	References: [Moore et al.(1993a)]					
NOTES:						

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species[Isotype]
259 G3-4	gp120(V2 170-180 BH10)	QKEYAFFYKLD	L	IIB gp120	murine(IgG _{2bκ})	

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Ho et al.(1991a), Ho et al.(1992), Fung et al.(1992), McKeating et al.(1992a), Moore & Ho(1993), Sullivan et al.(1993) , Sattentau et al.(1993) , Thali et al.(1993) , Moore et al.(1993a), Moore et al.(1994b), Gorni et al.(1994), Thali et al.(1994), Yoshiyama et al.(1994), Wu et al.(1995), Sattentau & Moore(1995), Jagodzinski et al.(1996), Moore & Sodroski(1996), Poignard et al.(1996a), Binley et al.(1997), Stamatatos et al.(1997), Ditzel et al.(1997)]

NOTES:

- G3-4: Also called G3.4
- G3-4: Binding is sensitive to removal of glycans by endo H – 50% neutralization of 4/9 primary isolates – has conformational features [Ho et al.(1991a)]
 - G3-4: Neutralizes IIB and RF, not MN – blocks sCD4-gp120, not as potent as MAb 15e – V2 binding MAbs BAT085 and G3-136 block G3-4 gp120 binding – sensitive to reduction of gp120 by DTT [Ho et al.(1992)]
 - G3-4: Substitutions in residues 176 to 184 affect MAb recognition – substitutions in V2 can result in gp120-gp41 dissociation [Sullivan et al.(1993)]
 - G3-4: Increased binding in the presence of sCD4 [Sattentau et al.(1993)]
 - G3-4: Conformational, does not bind well to denatured gp120 – not reactive with SF-2 gp120, and does not inhibit HIV-1 sera from binding to IIB gp120 [Moore & Ho(1993)]
 - G3-4: V2 region, marginal binding to peptide, binding inhibited by 183/184 PI/SG substitution [Moore et al.(1993a)]
 - G3-4: Conformationally sensitive – sporadic cross-reactivity among, and outside, B clade gp120s [Moore et al.(1994b)]
 - G3-4: Weakly neutralizing, IC₅₀
 - G3-4: gp41 mutation (582 A/T) that reduces neutralization of anti-CD4 binding site MAbs does not alter G3-4s ability to neutralize [Thali et al.(1994)]
 - G3-4: Neutralizes RF – substitutions 177 Y/H and 179 L/P in the V2 loop of RF reduce affinity and result in neutralization escape [Yoshiyama et al.(1994)]
 - G3-4: Reactive with BH10, RF, and MN – binds native, but not denatured or deglycosylated gp120, binds to deglycosylated V1V2 fusion protein, suggesting importance of glycans outside the V1V2 region [Wu et al.(1995)]
 - G3-4: Bound preferentially to the monomeric rather than oligomeric form of LAI gp120 – neutralizes Hx10 cell-free virus [Sattentau & Moore(1995)]
 - G3-4: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – deletion of the V3 loop from gp120 results in more potent G3-4 binding inhibition by CRDS – G3-4 epitope described as 176-184 FYKLDIPI and 191-193 YSL [Jagodzinski et al.(1996)]
 - G3-4: Binding enhanced by selected antibodies to C1, C4, C5, V3 and anti-CD4 binding site MAbs – enhances binding of selected V3, C4 and anti-CD4 binding site MAbs [Moore & Sodroski(1996)]
 - G3-4: Described epitope as STSIRGVKEYAFFYKLDI – binds oligomer – binding of V2 MAbs G3-136, G3-4 or BAT085 did not significantly alter gp120 dissociation from virus or expose the gp41 epitope of MAb 50-69, in contrast to anti-V3 MAbs [Poignard et al.(1996a)]
 - G3-4: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)]
 - G3-4: Called G3.4 – mediates gp120 virion dissociation in contrast to anti-V2 MAbs G3-136 – not neutralizing for SF162 or SF128A in either primary macrophages or PBMC [Stamatatos et al.(1997)]

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MAb ID	Location	WEAU	Sequence	Neutralizing	ImmunoGen	Species(Isotype)
260 BAT085	gp120(V2 170-180 IIIB)	gp120(175-184)	KEYAFFYKLD	L	Inact IIIB	murine(IgG ₁)

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Fung et al.(1987), Fung et al.(1992), Moore & Ho(1993), Pirofski et al.(1993), Thali et al.(1993), Moore et al.(1993a), D'Souza et al.(1994), Moore et al.(1994d), Gorny et al.(1994), Yoshiyama et al.(1994), Wu et al.(1995), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Binley et al.(1997), Ditzel et al.(1997)]

NOTES:

- BAT085: Also called BAT-085
- BAT085: V2 region – sCD4 does not block – neutralizes IIIB and some primary isolates, but not MN or RF – binds MN – deglycosylation or DDT reduction of gp120 does not diminish reactivity [Fung et al.(1992)]
- BAT085: Called BAT-85 – conformational, does not bind well to denatured gp120 – not reactive with SF-2 gp120, and does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)]
- BAT085: 7/8 V2 murine MAbs required gp120 native structure to bind, but BAT085 was the exception – type-specific [Moore et al.(1993a)]
- BAT085: Peptide affinities of G3-136 and G3-4 are 100-fold less than BAT085, but BAT085 has lower affinity for BH10 gp120 and is weaker at neutralization [Moore et al.(1993a)]
- BAT085: Multi-lab study for antibody characterization and assay comparison – did not bind MN or SF2 [D'Souza et al.(1994)]
- BAT085: Interacts with two overlapping peptides with region of overlap KEYAFFYKLD [Gorny et al.(1994)]
- BAT085: Neutralizes RF – substitution 177 Y/H in the V2 loop of RF does not inhibit neutralization, in contrast to MAbs G3-4 and SC258 [Yoshiyama et al.(1994)]
- BAT085: HXB10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu et al.(1995)]
- BAT085: Bound preferentially to the monomeric rather than oligomeric form of LAI gp120 – neutralizes cell free Hx10 [Sattentau & Moore(1995)]
- BAT085: Binding is blocked by other V2 region antibodies, enhanced by several anti-C1 MAbs, and anti-V3 MAb G511 – reciprocal enhancement of CD4i MAb 48d binding [Moore & Sodroski(1996)]
- BAT085: Epitope suggested to be QKEYAFFYKLD – binds oligomer – binding of V2 MAbs G3-136, G3-4 or BAT123 did not significantly alter gp120 dissociation from virus or expose the gp41 epitope of MAB 50-69, in contrast to anti-V3 MAbs [Poignard et al.(1996a)]
- BAT085: An antibody with moderate avidity as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)]

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
261 G3-136	gp120(V2 170-180 IIIB)	gp120(174-184)	QKEYAFFYKLD	L	purified IIIB gp120	murine(IgG)
Donor:	Tanox Biosystems Inc and David Ho, ADARC, NY					
References:	[Fung et al.(1992), Pirofski et al.(1993), Thali et al.(1993), Moore & Ho(1993), Moore et al.(1993a), Yoshiyama et al.(1994), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Binley et al.(1997), Stamatatos et al.(1997), Ditzel et al.(1997)]					
NOTES:						
	<ul style="list-style-type: none"> • G3-136: V2 region – binds and neutralizes IIIB and RF in CEM-SS cells, but not MN – neutralization activity against a few primary isolates in PBMC – sCD4 binding inhibits binding (contrast with BAT085) – deglycosylation or reduction of gp120 by DTT diminishes reactivity [Fung et al.(1992)] • G3-136: Conformational, does not bind well to denatured gp120 – not reactive with SF-2 gp120, and does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)] • G3-136: Marginal binding to peptide, binding inhibited by 183/184 PI/SG substitution [Moore et al.(1993a)] • G3-136: Binding enhanced by selected antibodies to C1, C4, C5, V3 and anti-CD4 binding site MAbs – enhances binding of selected V3, C4 and anti-CD4 binding site MAbs [Moore et al.(1993a)] • G3-136: HIV-1 RF V2 substitutions 177 Y/H and 179 L/P in the V2 loop of RF reduce affinity [Yoshiyama et al.(1994)] • G3-136: Bound preferentially to the monomeric rather than oligomeric form of LAI gp120 – neutralizes cell free Hx10 [Sattentau & Moore(1995)] • G3-136: Described epitope as STSIRGKVKEYAFFYKLDI – binds oligomer – binding of V2 MAbs G3-136, G3-4 or BAT123 did not significantly alter gp120 dissociation from virus or expose the gp41 epitope of MAb 50-69, in contrast to anti-V3 MAbs [Poignard et al.(1996a)] • G3-136: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)] • G3-136: Called G3.136 – does not mediate gp120 virion dissociation in contrast to anti-V2 MAb G3-4 – not neutralizing for SF162 or SF128A in either primary macrophages or PBMC [Stamatatos et al.(1997)] 					
262 38/12b	gp120(V2 172-191 HXB2)	gp120(176-195)	EYAFFYKLDIIPIDN-DTTSY	BH10 gp120	rat(unk)	
	References:	[Wu et al.(1995)]				
	<ul style="list-style-type: none"> • 38/12b: Broad specificity: HXB2, MN, SF162 – binds native and deglycosylated gp120 [Wu et al.(1995)] 					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
263 697-D	gp120(V2 161-180 IIIB)	gp120(165-184)	Conformational with weak reactivity to ISTSIRGKVQKEYAFFYKLD - neutralized 3/4 primary isolates, but none of 4 lab strains – V2 substitutions 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192-194 YSL/GSS abrogate binding – anti-C4 MAbs G3-536 and G45-60 enhance binding – mild oxidation of carbohydrate moieties inhibits binding [Gorny et al.(1994)]		HIV-1 infection	human(IgG _{1λ})
	Donor: Cellular Products Inc, Buffalo NY	FYKLD				
	References: [Gorny et al.(1994), Forthal et al.(1995), Moore & Ho(1995), Trkola et al.(1996a), Binley et al.(1997), Fouts et al.(1997)]					
	NOTES:					
	<ul style="list-style-type: none"> • 697-D: Also called 697D • 697-D: Conformational with weak reactivity to V2 peptide ISTSIRGKVQKEYAFFYKLD – neutralized 3/4 primary isolates, but none of 4 lab strains – V2 substitutions 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192-194 YSL/GSS abrogate binding – anti-C4 MAbs G3-536 and G45-60 enhance binding – mild oxidation of carbohydrate moieties inhibits binding [Gorny et al.(1994)] • 697-D: Not neutralizing, no ADCC activity, and no viral enhancing activity [Forthal et al.(1995)] • 697-D: Review: called 697/30D – neutralizes some primary, but not lab adapted strains [Moore & Ho(1995)] • 697-D: Partial inhibition of gp120 interaction with CCR-5 in a MIP-1$β$-CCR-5 competition study [Trkola et al.(1996a)] • 697-D: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)] • 697-D: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 697-D bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)] 					
264 12b	gp120(V2 162-181)	gp120(166-185)	STSIRGKVQKEYAFF- L (HXB10) YKLDI	BH10 rgp120	rat(IgG _{2a})	
	References: [Shotton et al.(1995), McKeating et al.(1996)]					
	NOTES:					
	<ul style="list-style-type: none"> • 12b: V2 MAb neutralized HXB2 – position 179-180 LD to DL abrogates binding – competes with 60b, but not 74 [Shotton et al.(1995)] • 12b: Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)] 					
265 38/60b	gp120(V2 172-191 HXB2)	gp120(176-195)	EYAFFYKLDIIPIDN- DTTSY	BH10 gp120	rat(unk)	
	References: [Wu et al.(1995)]					
	NOTES:					
	<ul style="list-style-type: none"> • 38/60b: Strain specificity: HXB2 – binds native and deglycosylated gp120 [Wu et al.(1995)] 					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
266 60b	gp120(V2 172-181 HXB2) References: [Shotton et al.(1995)]	gp120(176-185)	EYAFFYKLDI	N	BH10 rgp120	rat(IgG _{2b})
	NOTES:					
	• 60b: V2 MAb did not neutralize HXB2 – bound to rgp120 in ELISA – substitutions 179-180 LD/DL and 191-193 YSL/GSS abrogate binding, as do changes outside the minimum epitope – competes with 12b, but not 74 [Shotton et al.(1995)]					
267 74	gp120(V2 172-181) References: [Shotton et al.(1995)]	gp120(176-185)	EYAFFYKLDI	N	BH10 rgp120	rat(IgG ₁)
	NOTES:					
	• 74: V2 MAb did not neutralize HXB2 – did not bind rgp120 ELISA – position 179-180 LD to DL abrogates binding, as do changes outside the minimum epitope – does not compete with 60b or 12b, and is enhanced by two conformation dependent MAbs [Shotton et al.(1995)]					
268 3D3.B8	gp120(211-220 LAI) References: [Bohmstedt et al.(1990), Moore et al.(1994c)]	gp120(215-225)	EPIPIHYCAPA		Env glycopro	murine(IgG)
	NOTES:					
	• 3D3.B8: The relative affinity denatured/native gp120 is greater than 10 [Moore et al.(1994c)]					
269 4C11.D8	gp120(211-220 LAI) References: [Bohmstedt et al.(1990), Moore et al.(1994c)]	gp120(215-225)	EPIPIHYCAPA		Env glycopro	murine(IgM)
	NOTES:					
	• 4C11.D8: The relative affinity denatured/native gp120 is greater than 10 [Moore et al.(1994c)]					
270 322-151	gp120(201-220 LAI) Donor: G. Robey, Abbot Labs References: [Moore et al.(1994c), Moore et al.(1994d)]	gp120(215-225)	EPIPIHYCAPA		Env glycopro	murine(IgG)
	NOTES:					
	• 322-151: The relative affinity denatured/native gp120 is 30 [Moore et al.(1994c)]					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species/Isotype
271 110.1	gp120(200-217)	gp120(214-231)	PIPIHYCAPA ?	N	Env glycoprotein	human(unk)
	References: [Pincus et al.(1996), Valenzuela et al.(1998)]					
NOTES:						
	• 110.1: There is another antibody with this ID that binds to Env at positions 491-500 in LAI, see [Gosting et al.(1987)]					
	• 110.1: A panel immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus et al.(1996)]					
272 493-156	gp120(211-230 LAI)	gp120(215-234)	EPIPIHYCAPAGFAI-LKCNN	Env glycopro		murine(IgG)
	Donor: G. Robey, Abbot Labs					
	References: [Moore et al.(1994c)]					
NOTES:						
	• 493-156: The relative affinity denatured/native gp120 is >10 [Moore et al.(1994c)]					
273 GV4H3	gp120(219-226 IIIB)	(223-230)	APAGFAIL	gp120 complexed with mAb M77		murine(unk)
	References: [Denisova et al.(1996)]					
NOTES:						
	• GV4H3: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes					
274 J1	gp120(222-231 LAI)	gp120(226-235)	GFAIIKCNNK	peptide		murine(IgG1)
	Donor: J. Hoxie, U. Penn.					
	References: [Moore et al.(1994c), Moore et al.(1994d), Cook et al.(1994)]					
NOTES:						
	• J1: The relative affinity denatured/native gp120 is 30 [Moore et al.(1994c)]					
	• J1: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)]					
275 J3	gp120(222-231 LAI)	gp120(226-235)	GFAIIKCNNK	peptide		murine(IgG1)
	Donor: J. Hoxie, U. Penn.					
	References: [Moore et al.(1994c), Cook et al.(1994)]					
NOTES:						
	• J3: The relative affinity denatured/native gp120 is 30 [Moore et al.(1994c)]					
	• J3: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)]					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
276 MF87.1	gp120(242-261 LAI)	gp120(256-265)	RPVVSTQLLL	Env		murine(IgG)
	References: [Thiria et al.(1989), Moore et al.(1994c)]					
NOTES:	• MF87.1: The relative affinity denatured/native gp120 is 10 – mutations 252 R/W, 257 T/G, and 257 T/R impair binding [Moore et al.(1994c)]					
277 MF169.1	gp120(242-261 LAI)	gp120(256-265)	RPVVSTQLLL	Env		murine(IgG)
	References: [Thiria et al.(1989), Moore et al.(1994c), Moore et al.(1994d)]					
NOTES:	• MF169.1: The relative affinity denatured/native gp120 is 11 – mutations 252 R/W, 257 T/G, and 257 T/R impair binding [Moore et al.(1994c)]					
278 MF170.1	gp120(242-261 LAI)	gp120(256-265)	RPVVSTQLLL	Env		murine(IgG)
	References: [Thiria et al.(1989), Moore et al.(1994c), Moore et al.(1994d)]					
NOTES:	• MF170.1: The relative affinity denatured/native gp120 is 15 – mutations 252 R/W, 257 T/G, and 257 T/R impair binding to denatured and native gp120, and 262N/T, 269 E/L and 281 A/V to only native gp120 [Moore et al.(1994c)]					
279 213.1	gp120(242-261 LAI)	gp120(256-265)	RPVVSTQLLL	Env glycopro		murine(IgG1)
	Donor: Claudine Bruck					
	References: [Thiria et al.(1989), Moore & Ho(1993), Moore et al.(1994c)]					
NOTES:	• 213.1: Bound preferentially to denatured IIIB and SF2 gp120 [Moore & Ho(1993)] • 213.1: The relative affinity denatured/native gp120 is 100 – mutations 252 R/W, 257 T/G or T/R impair binding [Moore et al.(1994c)] • 213.1: UK Medical Research Council AIDS reagent: ARP334					
280 M89	gp120(C2 252-271 LAI)	gp120(256-275)	RPVVSTQLLNGSLA- EEEVV	N	451 Env	murine(IgG1)
	Donor: Fulvia di Marzo Veronese					
	References: [di Marzo Veronese et al.(1992), Moore et al.(1994c), Moore et al.(1994d)]					
NOTES:	• M89: Immunoblot reactive, RIP negative, for strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese et al.(1992)] • M89: C2 region – the relative affinity for denatured/native gp120 is >30 – mutations 257 T/R and 269 E/L impair binding [Moore et al.(1994c)]					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
281 B12	gp120(C2 252-271 LAI)	gp120(256-275)	RPVVSTQ LLLNGSLA- EEEVV	mis-folded LAI rgp160	murine(IgG)	
	References: [Moore et al.(1994c)]					
	NOTES:					
	• B12: C2 region – the relative affinity for denatured/native gp120 is 27 – mutations 257 T/R and 262 N/T impair binding [Moore et al.(1994c)]					
282 B13	gp120(C2 252-271 LAI)	gp120(256-275)	RPVVSTQ LLLNGSLA- EEEVV	mis-folded LAI rgp160	murine(IgG _{2a})	
	Donor: George Lewis					
	References: [Moore & Ho(1993), Moore et al.(1994c), Abacioglu et al.(1994), Moore et al.(1994d), Pincus et al.(1996)]					
	NOTES:					
	• B13: Also called Bh13					
	• B13: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]					
	• B13: the relative affinity for denatured/native gp120 is 30 – mutations 257 T/R and 269 E/L impair binding [Moore et al.(1994c)]					
	• B13: C2 region – epitope boundaries mapped by peptide scanning, core epitope: TQLLN [Abacioglu et al.(1994)]					
	• B13: Called Bh13 – binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing [Pincus et al.(1996)]					
283 B24	gp120(C2 257-262 BH10)	gp120(261-266)	TQLLN	mis-folded LAI rgp160	murine(IgG _{2a})	
	References: [Abacioglu et al.(1994)]					
	NOTES:					
	• B24: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					
284 B3	gp120(C2 257-262 BH10)	gp120(261-266)	TQLLN	mis-folded LAI rgp160	murine(IgG ₁)	
	References: [Abacioglu et al.(1994)]					
	NOTES:					
	• B3: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
285 B21	gp120(C2 257-262 BH10)	gp120(261-266)	TQLLLN	mis-folded LAI rgp160	murine(IgG1)	
	References: [Abacioglu et al.(1994)]					
	NOTES:					
	• B21: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					
286 B23	gp120(C2 257-262 BH10)	gp120(261-266)	TQLLLN	mis-folded LAI rgp160	murine(IgG _{2a})	
	References: [Abacioglu et al.(1994)]					
	NOTES:					
	• B23: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					
287 B25	gp120(C2 257-262 BH10)	gp120(261-266)	TQLLLN	mis-folded LAI rgp160	murine(IgG1)	
	References: [Abacioglu et al.(1994)]					
	NOTES:					
	• B25: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					
288 B29	gp120(C2 257-263 BH10)	gp120(261-267)	TQLLLNG	mis-folded LAI rgp160	murine(IgG _{2a})	
	References: [Abacioglu et al.(1994)]					
	NOTES:					
	• B29: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					
289 B26	gp120(C2 257-263 BH10)	gp120(261-267)	TQLLLNG	mis-folded LAI rgp160	murine(IgG1)	
	References: [Abacioglu et al.(1994)]					
	NOTES:					
	• B26: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					
290 B36	gp120(C2 257-263 BH10)	gp120(261-267)	TQLLLNG	mis-folded LAI rgp160	murine(IgG1)	
	References: [Abacioglu et al.(1994)]					
	NOTES:					
	• B36: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)]					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
291 C13	gp120(C2 252-271 LAI)	gp120(256-275)	RPVVSTQLLL-NGSLAAEEEVV	mis-folded LAI rgp160	murine(IgG ₁)	
	Donor: George Lewis References: [Moore & Ho(1993), Moore et al.(1994c), Abacioglu et al.(1994)]					
	NOTES:					
	• C13: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]					
	• C13: The relative affinity for denatured/native gp120 is 36 – mutations 257 T/R, 267 E/L, and 269 E/L impair binding [Moore et al. (1994c)]					
	• C13: epitope boundary extended to RPVVSTQLLLNGSLAAEEEVVIR, to take into account the effect of a point mutation [Abacioglu et al. (1994)]					
	• C13: NIH AIDS Research and Reference Reagent Program: 1209					
292 110.E	gp120(C2 262-281 LAI)	gp120(266-285)	NGSLAAEEEVV-IRSVNFTTDNA	Env glycopro	murine(IgG)	
	Donor: F. Traincard References: [Moore et al.(1994c), Moore et al.(1994d)]					
	NOTES:					
	• 110.E: The relative affinity for denatured/native gp120 is 7.3 [Moore et al.(1994c)]					
293 110.C	gp120(C2 261-280 LAI)	gp120(275-284)	VIRSVNFTDN	Env glycopro	murine(IgG)	
	Donor: F. Traincard, Hydridolabs, Institut Pasteur References: [Moore et al.(1994c), Moore et al.(1994d), Valenzuela et al.(1998)]					
	NOTES:					
	• 110.C: The relative affinity for denatured/native gp120 is 1 [Moore et al.(1994c)]					
	• 110.C: Only slightly reduces LAI viral binding or entry into CEM cells [Valenzuela et al.(1998)]					
294 IIIB-V3-21	gp120(V3 299-304 IIIB)	gp120(298-303)	INCTRTP	N	Peptide	murine(IgG ₁)
	Donor: J. Laman References: [Laman et al.(1992), Laman et al.(1993), Valenzuela et al.(1998)]					
	NOTES:					
	• IIIB-V3-21: Also called V3-21					
	• IIIB-V3-21: Binds to the base of the V3 loop on denatured gp120 [Laman et al.(1992)]					
	• IIIB-V3-21: Binds to NP40 treated gp120, and epitope is probably obscured by local glycosylation [Laman et al.(1993)]					
	• IIIB-V3-21: Does not block HIV-1 LAI binding or entry into CEM cells [Valenzuela et al.(1998)]					
	• IIIB-V3-21: UK Medical Research Council AIDS reagent: ARP3048					
	• IIIB-V3-21: NHH AIDS Research and Reference Reagent Program: 1725					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
295 IIIB-V3-26	gp120(V3 299-304 IIIB)	gp120(295-311)	SVEINCTRPN- NNTRKSI	N	Peptide	murine(IgG ₁)
	References: [Laman et al.(1992)]					
	NOTES:					
	• IIIB-V3-26: Binds to the base of the V3 loop on denatured gp120 [Laman et al.(1992)]					
296 MO97/V3	gp120(V3 299-308 IIIB)	gp120(303-312)	PNNNTRKSIR	N	rpB1 (IIIB Env 286-467)	human(IgM)
	References: [Ohlin et al.(1992)]					
	NOTES:					
	• MO97/V3: MO97: Generated through <i>in vitro</i> "immunization" of uninfected-donor lymphocytes [Ohlin et al.(1992)]					
297 8/38c	gp120(V3 300-315 HXB10)	gp120(304-317)	NNNTRKIRI- QRGPGR	L	rBHI0 gp120	rat(IgG _{2a})
	Donor: C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK					
	References: [McKeating et al.(1992a), Sattentau & Moore(1995), Jeffs et al.(1996)]					
	NOTES:					
	• 8/38c: Also called 8/38/1c					
	• 8/38c: Binds to virion gp120 and neutralizes only in the presence of sCD4 [McKeating et al.(1992a)]					
	• 8/38c: Binds equally well to monomer and oligomer, less rapid association rate than other anti-V3 antibodies, and an associated less potent neutralization of lab strains [Sattentau & Moore(1995)]					
	• 8/38c: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs et al.(1996)]					
	• 8/38c: UK Medical Research Council AIDS reagent: ARP3039					
298 8/64b	gp120(V3 300-315 HXB10)	gp120(304-317)	NNNTRKIRI- QRGPGR	L	rBHI0 gp120	rat(IgM)
	References: [McKeating et al.(1992a)]					
	NOTES:					
	• 8/64b: Binds to virion gp120 and neutralizes only in the presence of sCD4 [McKeating et al.(1992a)]					
	• 8/64b: UK Medical Research Council AIDS reagent: ARP3036					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
299 polyclonal	gp120(V3 IIIB)	gp120(305-328)	NNTRKSIRIQRGP-GRAFVTIGKIGN	L	oral immunization – peptide plus cholera toxin adjuvant	murine(IgA)
References: [Bukawa et al.(1995)]						
NOTES:						
• polyclonal: Polyclonal secretory IgA antibody raised by mucosal immunization is able to neutralize IIIB, SF2, and MN – HIV-1 neutralization may be due to V3, CD4 or HPG30 component of the multicomponent peptide immunogen [Bukawa et al.(1995)]						
300 polyclonal	gp120(V3 IIIB)	gp120(305-325)	CNNTRKSIRIQRGP-GRAFVTIGK	L	?	Guinea pig IgG
Donor: D. Bolognesi and T. Matthews, Duke University						
References: [Allaway et al.(1993)]						
NOTES:						
• polyclonal: Synergy with combinations of CD4-based molecules in inhibition of HIV-1 Env mediated cell fusion [Allaway et al.(1993)]						
301 MO99/V3	gp120(V3 304-308 IIIB)	gp120(308-312)	RKSIR	N	rpB1 (IIIB Env 286-467)	human(IgM)
References: [Ohlin et al.(1992)]						
NOTES:						
• MO99/V3: MO99: Generated through <i>in vitro</i> “immunization” of uninfected-donor lymphocytes [Ohlin et al.(1992)]						
302 DO 142-10	gp120(V3 MN)	gp120(309-322)	KRIHIGPGRAYTT	HIV-1 infection	human(unk)	
References: [Seligman et al.(1996)]						
NOTES:						
• DO 142-10: Fab fragment; Competition ELISAs with serial deletions defined the epitope KRIHIGPGRAYTT [Seligman et al.(1996)]						
303 TH1	gp120(V3)	(unk)	unk	L (MN,JRCSF)	human(IgG _{1,λ})	
Donor: Michael Fung, Tanox Biosystem, USA						
References: [D'Souza et al.(1995)]						
NOTES:						
• TH1: Found to neutralize MN and JRCSF, but not two B subtype primary isolates, nor a D subtype primary isolate, by most labs in a multi-laboratory study involving 11 labs [D'Souza et al.(1995)]						

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
304 D47	(V3 IIIB)	(unk)	unk	IIIIB vaccinia expressed Env		murine(unk)
	References: [Richardson Jr et al.(1996)]					
	NOTES:					
	• D47: Used for capture of oligomeric Env for antigen capture ELISA – binding of this antibody to oligomeric Env IIIIB was not blocked by human sera from the US, consistent with a low prevalence of IIIB-like V3 strains [Richardson Jr et al.(1996)]					
305 F19.48-3	gp120(V3 312-324 LAI)	gp120(309-321)	IRIQRGPGRAFVT	L	IIIIB rgp120 294-474	murine(IgG _{2aκ})
	References: [Boudet et al.(1994)]					
	NOTES:					
	• F19.48-3: Strain specific – used to raise anti-idiotypic antibodies [Boudet et al.(1994)]					
306 F19.26-4	gp120(V3 312-324 LAI)	gp120(309-321)	IRIQRGPGRAFVT	L	IIIIB rgp120 294-474	murine(IgG _{2aκ})
	References: [Boudet et al.(1994)]					
	NOTES:					
	• F19.26-4: Strain specific – used to raise anti-idiotypic antibodies [Boudet et al.(1994)]					
307 F19.57-11	gp120(V3 312-324 LAI)	gp120(309-321)	IRIQRGPGRAFVT	L (LAI)	IIIIB rgp120 294-474	murine(IgG _{1κ})
	References: [Boudet et al.(1991), Boudet et al.(1994), Boudet et al.(1995)]					
	NOTES:					
	• F19.57-11: MAb F19.57-11 is strain specific for LAI – used to raise anti-idiotypic rabbit antibodies (called 57-B Ab2) [Boudet et al.(1994)]					
	• F19.57-11: Anti-anti-idiotypic antibodies (Ab3) were raised in BALB/c mice that had greater breadth of reactivity than the original F19.57-11 (Ab3 could also recognize 1282 and SF2, with aa TRK(R or S)IYIGPGRA(WY or FH)T) [Boudet et al.(1995)]					
308 M096/V3	gp120(309- 318 + 329-338)	gp120	IQRGPGRAFV + AHCNISRAKW	rIIIIB Env 286-467	human(IgM)	
	References: [Ohlin et al.(1992)]					
	NOTES:					
	• M096/V3: M096: Generated through <i>in vitro</i> “immunization” of uninfected-donor lymphocytes [Ohlin et al.(1992)]					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunocon-	Species(Isootype)
309 MO101/V3,C4	gp120(314-323 + 494-503)	gp120	GRAFVTIGK + LGVAPTKAKR	rIIIB Env 286-467	human(IgM)	
	References: [Ohlin et al.(1992)]					
	NOTES:					
	• MO101/V3,C4: MO101: generated through <i>in vitro</i> "immunization" of uninfected-donor lymphocytes – reacts with peptides from the V3 and C4 regions [Ohlin et al.(1992)]					
310 N70-1.9b	gp120(V3 316-322) gp120(315-320)	PGRAFY	L	HIV-1 infection	human(IgG1)	
	References: [Robinson et al.(1990), Scott Jr et al.(1990)]					
	NOTES:					
	• N70-1.9b: Type specificity [Robinson et al.(1990)]					
	• N70-1.9b: Type specific neutralization, ADCC directed against MN infected cells [Scott Jr et al.(1990)]					
311 MAG 49	gp120(V3 302-321 BH10)	gp120(306-323) FVTIG	NTRKSIRIQRGPGRAL	L	sCD4-(rHXB2 gp120)-complex	murine(unk)
	References: [Kang et al.(1994), Moore & Sodroski(1996)]					
	NOTES:					
	• MAG 49: Binds a V3 loop peptide – sensitive to both V3 loop mutations and a mutation at the base of the V1/V2 loop structure (120/121 VK/LE) [Kang et al.(1994)]					
	• MAG 49: Called #49 in this text. Binding enhanced by anti-C1 MAbs 133/290, 135/9, and by many anti-CD4 binding site MAbs – reciprocal enhancement of some anti-V2 MAbs – reciprocal binding inhibition of anti-V3 MAbs [Moore & Sodroski(1996)]					
312 MAG 53	gp120(V3 302-321 BH10)	gp120(306-323) FVTIG	NTRKSIRIQRGPGRAL	L	sCD4-(rHXB2 gp120)-complex	murine(unk)
	References: [Kang et al.(1994)]					
	NOTES:					
	• MAG 53: Binds a V3 loop peptide – sensitive to both V3 loop mutations and a mutation at the base of the V1/V2 loop structure (120/121 VK/LE) [Kang et al.(1994)]					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
313 MAG 56	gp120(V3 302-321 BH10)	gp120(306-323)	NTRKSIRIQRGPGRA-FVTIG	L	sCD4-(rHXB2 gp120)-complex	murine(unk)
	References: [Kang et al.(1994)]					
	NOTES:					
	• MAG 56: Binds a V3 loop peptide – sensitive to both V3 loop mutations and a mutation at the base of the V1/V2 loop structure (120/121 VK/LE) [Kang et al.(1994)]					
314 MAG 109	gp120(V3 302-321 BH10)	gp120(306-323)	NTRKSIRIQRGPGRA-FVTIG	L	sCD4-(rHXB2 gp120)-complex	murine(unk)
	References: [Kang et al.(1994)]					
	NOTES:					
	• MAG 109: Binds a V3 loop peptide – sensitive to both V3 loop mutations and a mutation at the base of the V1/V2 loop structure (120/121 VK/LE) [Kang et al.(1994)]					
315 polyclonal	gp120(V3 306-338 BH10)	gp120(303-334)	PNNNTRKSIRIQRGPGRAFVTIGKIGNMIRQ-AHC	L	Peptide	rabbit(IgG)
	References: [Neurath & Strick(1990)]					
	NOTES:					
	• polyclonal: 21 V3 loop variant peptides spanning this region were tested and serological cross-reactivity correlated with divergence [Neurath & Strick(1990)]					
316 1026	gp120(V3 tip MN)	gp120(314-319)	close to GPGRAF ?	L	rgp120 MN	murine(IgG)
	References: [Nakamura et al.(1993), Bou-Habib et al.(1994)]					
	NOTES:					
	• 1026: Bound diverse strains, neutralizing activity against MN [Nakamura et al.(1993)]					
	• 1026: Greater affinity for T cell-tropic strain T-CSF, derived from JR-CSF, than to the primary isolate JR-CSF [Bou-Habib et al.(1994)]					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunocongen	Species(Isootype)
317 9284	gp120(V3 307-318 IIIB)	gp120(305-316) NNTRKSIRIQRG	L	disrupted IIIB virion	murine(IgG1)	

Donor: Dupont de Nemours, Les Ulis, France or Wilmington, Delaware

References: [Skinner et al.(1988b), Skinner et al.(1988a), Sattentau & Moore(1991), Wyatt et al.(1992), McKeating et al.(1992a), Sattentau et al.(1993), Moore et al.(1993b), Trujillo et al.(1993), Thali et al.(1993), VanCott et al.(1994), Thali et al.(1994), Cook et al.(1994), Okada et al.(1994), Sorensen et al.(1994), Sattentau & Moore(1995), VanCott et al.(1995), Fontenot et al.(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Binley et al.(1997)]

NOTES:

- 9284: IIIB type-specific binding and neutralization [Skinner et al.(1988b)]
- 9284: Two fold increase in binding to gp120 in the presence of bound sCD4 [Sattentau & Moore(1991)]
- 9284: Single amino acid substitutions in the C4 region (427 W/V or W/S) or at the base of the V3 loop (298 R/G) can significantly increase binding and neutralization– position 427 is also important for CD4 binding and anti-CD4 binding site MAbs [Wyatt et al.(1992)]
- 9284: Increased binding in the presence of sCD4 [Sattentau et al.(1993)]
- 9284: Inhibits C4 region antibodies (G3-299, G3-519) which have conformational requirements [Moore et al.(1993b)]
- 9284: Peptide RIQRGPGRAFVTIGKIGNMRQA – Reacts with three human brain proteins of 35, 55, 110 kDa – called NEA-9284 [Trujillo et al.(1993)]
- 9284: Does not bind MN gp120, just IIIB [VanCott et al.(1994)]
- 9284: gp41 mutation that confers resistance to neutralization by anti-CD4 binding site antibodies does not reduce neutralizing efficiency of this V3 region MAb [Thali et al.(1994)]
- 9284: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer *in vitro* [Cook et al.(1994)]
- 9284: Binding domain aa 301-310: TRKSIRIQRG – mutations in the V3 loop from basic residues can destroy virus infectivity and syncytium formation: 306 R/T, 309 R/T and 313 R/G can also reduce binding of V3 MAbs with two different binding sites: 9284 and 0.5β – called NEA9284 [Okada et al.(1994)]
- 9284: Did not neutralize infection of HIV/HTLV-I pseudotype [Sorensen et al.(1994)]
- 9284: Binds equally well to monomer and oligomer, rapid association and potent neutralization of lab strains – neutralizes cell-free virus Hx10 [Sattentau & Moore(1995)]
- 9284: Used to monitor HIV-1 Env expression in infected H9 cells, binds native and reduced gp120s similarly [VanCott et al.(1995)]
- 9284: Binds V3 loop – anti-C1 MAbs 133/290 and 135/9 enhance binding – reciprocal binding inhibition of other anti-V3 MAbs [Moore & Sodroski(1996)]
- 9284: V3 MAbs 9284, BAT123, 110.5, and 110.1 could each significantly increase gp120 dissociation from virus, mimicking sCD4, and expose the gp41 epitope for MAb 50-69, in contrast to anti-V2 MAbs [Poignard et al.(1996a)]
- 9284: A high avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)]

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunoigen	Species (Isotype)
318 1034	gp120(V3 tip MN) References: [Bou-Habib et al.(1994)]	gp120(314-319) close to GPGRAF ?	L	rgp120 MN		murine(IgG)
NOTES:	• 1034: Greater affinity for T cell tropic T-CSF, derived from JR-CSF, than to the primary isolate JR-CSF [Bou-Habib et al.(1994)]					
319 polyclonal	gp120(V3 304-318 LAI) References: [Chin et al.(1995)]	gp120(306-320) RKSTRIQRGPGRAFV	?		human(IgG, IgM)	
NOTES:	• polyclonal: Mimicking the humoral immune response <i>in vitro</i> supports isotype switching – human IgG MAbs were generated from naive donors [Chin et al.(1995)]					
320 Aw	gp120(V3 tip, Gun-1wt) References: [McKnight et al.(1995)]	gp120(309-322) KSITIGPGRFAFHAI	L	V3 peptide		rat(unk)
NOTES:	• Aw: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – Aw gives weak neutralization both wt and v strains [McKnight et al.(1995)]					
321 Bw	gp120(V3 tip, Gun-1wt) References: [McKnight et al.(1995)]	gp120(309-322) KSITIGPGRFAFHAI	L	V3 peptide		rat(unk)
NOTES:	• Bw: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – Bw gives weak neutralization of only the wt strain, does not bind to variant [McKnight et al.(1995)]					
322 Dv	gp120(V3 tip, Gun-1v) References: [McKnight et al.(1995)]	gp120(309-322) KSITIGSGRAFHAI	L	V3 peptide		rat(unk)
NOTES:	• Dv: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight et al.(1995)]					
323 Fv	gp120(V3 tip, Gun-1v) References: [McKnight et al.(1995)]	gp120(309-322) KSITIGSGRAFHAI	L	V3 peptide		rat(unk)
NOTES:	• Fv: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight et al.(1995)]					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunoigen	Species (Isotype)
324 Gv	gp120(V3 tip, Gun-1v)	gp120(309-322)	KSTIGSGRAFHAI	L	V3 peptide	rat(unk)
	References: [McKnight et al.(1995)]					
NOTES:						
	• Gv: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight et al.(1995)]					
325 Hv	gp120(V3 tip, Gun-1v)	gp120(309-322)	KSTIGSGRAFHAI	L	V3 peptide	rat(unk)
	References: [McKnight et al.(1995)]					
NOTES:						
	• Hv: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight et al.(1995)]					
326 polyclonal	gp120(V3 304-318 LAI)	gp120(310-321)	RHIGPGRAFYT	?		human(IgG, IgM)
	References: [Langedijk et al.(1995)]					
NOTES:						
	• polyclonal: Polyclonal sera from six individuals tested for reactivity against a panel of peptides based on autologous sequences provide evidence for immunological escape mutations in the tip of the V3 loop [Langedijk et al.(1995)]					
327 C311E	gp120 (V3 309-316 MN)	gp120(308-315)	RKRRHIGP	L	HIV infection	chimpanzee
	References: [Warriner et al.(1996)]					
NOTES:						
	• C311E: Synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G [Warriner et al.(1996)]					
328 5G11	gp120(V3 loop)	gp120	?	?		(unk)
	Donor: S. Nigida and L. Arthur, NCI, Frederick, MD USA					
References: [Moore & Sodroski(1996)]						
NOTES:						
	• 5G11: Binds to conformation sensitive epitope in the V3 loop – reciprocal inhibition of other V3 loop MAbs – reciprocal enhancement of some C1-C5 MAbs (unusual for an anti-V3 MAb) and CD4 binding site MAbs – and enhances binding of V2 MAbs [Moore & Sodroski(1996)]					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species (Isotype)
329 110.3	gp120(V3 308-328 BRU)	sp120(312-319)	QRGPGRAF	L	BRU infected cell lysates	murine(IgG _{1κ})
	References: [Kinney Thomas et al.(1988), Evans et al.(1989), Langedijk et al.(1992), Pirofski et al.(1993), Connally et al.(1994)]					
	NOTES:					
	<ul style="list-style-type: none"> • 110.3: Included as a control [Evans et al.(1989)] • 110.3: MAb variable region sequenced – heavy chain: V 7138(40), D deletion, J_H4 – light chain: V_κ21(47), J_κ2 [Pirofski et al.(1993)] • 110.3: An anti-idiotypic MAb generated against 110.3 both mimics and binds to V3, suggesting that the V3 loop may associated with itself [Connally et al.(1994)] 					
330 110.4	gp120(V3 308-328 BRU)	sp120(312-319)	QRGPGRAF	L	BRU infected cell lysates	murine(IgG _{1κ})
	Donor: Genetic Systems Corp, Seattle WA, E. Kinney-Thomas					
	References: [Kinney Thomas et al.(1988), Thali et al.(1992b), Langedijk et al.(1992), Thali et al.(1993), Pirofski et al.(1993), Arendrup et al.(1993), Thali et al.(1994), Boudet et al.(1994), Connally et al.(1994), McDougal et al.(1996), Valenzuela et al.(1998)]					
	NOTES:					
	<ul style="list-style-type: none"> • 110.4: 313 P/S substitution in the V3 region disrupts binding [Thali et al.(1992b)] • 110.4: MAb variable region sequenced – heavy chain: V 3660-SB32, D closest to DSP2.3, 2.4 and .6, J_H2 – light chain: V_κ21, J_κ2 [Pirofski et al.(1993)] • 110.4: Primary isolates from different time points from one individual were not susceptible to neutralization by 110.4 [Arendrup et al.(1993)] • 110.4: gp41 mutation that confers resistance to neutralization by anti-CD4 binding site antibodies does not reduce neutralizing efficiency of this V3 region MAb [Thali et al.(1994)] • 110.4: An anti-idiotypic MAb generated against 110.3 also blocks binding of 110.4 [Connally et al.(1994)] • 110.4: Neutralization of LAI in CEM cells by anti-V3 MAbs 110.4 and N11-20 is through inhibition of viral binding to the cell [Valenzuela et al.(1998)] • 110.4: Neutralizes HIV-1 LAI [McDougal et al.(1996)] 					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immuno	Species (Isotype)
331 110.5	gp120(V3 308-328 BRU)	sp120(312-319)	QRGPGRAF	L	BRU infected cell lysates	murine(IgG1 κ)

Donor: E. Kinney-Thomas or Genetic Systems, Seattle WA

References: [Kinney Thomas et al.(1988), Moore et al.(1990), Cordell et al.(1991), Sattentau & Moore(1991), Langedijk et al.(1992), McKeating et al.(1992a), Pirofski et al.(1993), Moore et al.(1993b), Thali et al.(1993), Klasse et al.(1993a), Sattentau et al.(1995), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), McDougal et al.(1996), Jeffs et al.(1996), Binley et al.(1997), Ugolini et al.(1997)]

NOTES:

- 110.5: Did not induce dissociation of gp120, as sCD4 did – discrepancy with [Poignard et al.(1996a)], that was suggested to be due to MAbs interference with detection, as the gp120-MAb complex was denatured in the Poignard study [Moore et al.(1990)]
- 110.5: Binding insensitive to gp120 reduction [Cordell et al.(1991)]
- 110.5: Two fold increase in binding to gp120 in the presence of bound sCD4 [Sattentau & Moore(1991)]
- 110.5: Variable region sequenced – heavy chain: V 3660-SB32, D closest to DSP2.3, 2.4 and .6, J_H2 – light chain: V_K21, J_K2 [Pirofski et al.(1993)]
- 110.5: Thrombin cleavage of V3 loop between R-315 and A-316 abrogates binding – can inhibit C4 region antibody which has conformational requirements (G3-299) – binding to native gp120 100-300 fold greater than to denatured [Moore et al.(1993b)]
- 110.5: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to conformationally sensitive neutralizing MAbs – neutralization efficiency of 110.5 is not affected [Reitz Jr. et al.(1988), Klasse et al.(1993a)]
- 110.5: Pretreatment of HX10-infected H9 cells with sCD4 decreases signal from 110.5 at 37 degrees due to dissociation of gp120-gp41 [Sattentau et al.(1995)]
- 110.5: Binds with high affinity to monomer and oligomer, rapid association and potent neutralization of lab strains – neutralizes cell-free Hx10 [Sattentau & Moore(1995)]
- 110.5: Reciprocal binding inhibition with other anti-V3 MAbs [Moore & Sodroski(1996)]
- 110.5: V3 MAbs 9284, BAT123, 110.5, and 110.I could each significantly increase gp120 dissociation from virus, mimicking sCD4, and expose the gp41 epitope for MAB 50-69, in contrast to anti-V2 MAbs [Poignard et al.(1996a)]
- 110.5: Neutralizes HIV-1 LAI [McDougal et al.(1996)]
- 110.5: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs et al.(1996)]
- 110.5: A high avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)]
- 110.5: Viral binding inhibition by 110.5 was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997);]

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
332 5023A	gp120(V3 311-317 BH10)	gp120(313-319)	RgPGRAF	L	15 mer synthetic BH10 V3 peptide	murine(IgG)
Donor: Paul Durdla, Du Pont de Nemours and Co						
References: [Langedijk et al.(1991), D'Souza et al.(1991), Back et al.(1993), Rovinski et al.(1995)]						
NOTES:						
• 5023A: Generation and Fine mapping of murine MAbs [Langedijk et al.(1991)]						
• 5023A: Called 5023 – Langedijk also has an MAb called 5023B – strong cross-reactive neutralizing MAb [D'Souza et al.(1991)]						
• 5023A: Called 5023 – Langedijk also has an MAb called 5023B – gp41 amino acid substitutions 668 (N/S) and 675 (I/M) in gp41 interfere with 5023s neutralization potency, region 662-675 is ELDKWANLWNWFNI [Back et al.(1993)]						
• 5023A: Called 5023 in this paper – Used to precipitate gp160 in immunoblots in a study examining the feasibility of using unprocessed gp160 glycoprotein as an immunogen [Rovinski et al.(1995)]						
333 178.1	gp120(V3 305-309 BH10)	gp120(309-313)	KSIRI	L	yeast gp160 IIIB	murine(IgG _{2a})
Donor: C. Thiriait, Smith Kline and MRC AIDS reagent project						
References: [Thiriait et al.(1989), Back et al.(1993), Moore & Ho(1993), Cook et al.(1994)]						
NOTES:						
• 178.1: reacts to gp120 and gp160 in RIPA ELA and immunoblot [Thiriait et al.(1989)]						
• 178.1: Called 178.1.1 – conformational, does not bind well to denatured gp120 [Moore & Ho(1993)]						
• 178.1: gp41 amino acid substitutions 668 (N/S) and 675 (I/M) in gp41 interfere with 5023s neutralization potency, region 662-675 is ELDKWANLWNWFNI [Back et al.(1993)]						
• 178.1: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer <i>in vitro</i> – binding of GalCer to gp120 inhibited but did not completely block MAb binding[Cook et al.(1994)]						
• 178.1: UK Medical Research Council AIDS reagent: ARP331						
334 5042A	gp120(V3 310-315 BH10)	gp120(312-317)	QrGPGR	L	15 mer synthetic BH10 V3 peptide	murine(IgG)
References: [Langedijk et al.(1991)]						
NOTES:						
• 5042A: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)]						

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
335 5025A	gp120(V3 313-317 BH10)	gp120(315-319)	pgRRAF	L	15 mer synthetic BH10 V3 peptide	murine(IgG)
	Donor: Paul Durdla, Du Pont de Nemours and Co					
	References: [Langedijk et al.(1991), D'Souza et al.(1991)]					
	NOTES:					
	• 5025A: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)]					
	• 5025A: 5025: Called 5025 – strain specific weakly neutralizing [D'Souza et al.(1991)]					
336 5020	gp120(V3 311-316 BH10)	gp120(313-318)	RGGRA	N	15 mer synthetic BH10 V3 peptide	murine(IgG)
	References: [Langedijk et al.(1991)]					
	NOTES:					
	• 5020: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)]					
337 5042B	gp120(V3 310-315 BH10)	gp120(312-317)	QRGPGr	N	15 mer synthetic BH10 V3 peptide	murine(IgG)
	References: [Langedijk et al.(1991)]					
	NOTES:					
	• 5042B: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)]					
338 5025B	gp120(V3 310-316 BH10)	gp120(312-318)	QRGPGrA	N	15 mer synthetic BH10 V3 peptide	murine(IgG)
	References: [Langedijk et al.(1991)]					
	NOTES:					
	• 5025B: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)]					
339 5023B	gp120(V3 309-316 BH10)	gp120(311-318)	IQRGPGrA	N	15 mer synthetic BH10 V3 peptide	murine(IgG)
	References: [Langedijk et al.(1991)]					
	NOTES:					
	• 5023B: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)]					

Mab ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
340 110.I	gp120(V3 316-322)	gp120(318-324)	AFVTIGK	L	recombinant gp120	murine(unk)
Donor:	F. Traincard, Pasteur Institute, France					
References:	[Moore et al.(1993b), Moore et al.(1994c), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a)]					
NOTES:						
	<ul style="list-style-type: none"> • 110.I: Binds to carboxy-terminal side of the V3 loop – inhibits binding of C4 region MAb G3-299 [Moore et al.(1993b)] • 110.I: Binds equally well to monomer and oligomer, rapid association and potent neutralization of lab strains [Sattentau & Moore(1995)] • 110.I: Reciprocal binding inhibition with other anti-V3 and anti-C4 MAbs – and enhances binding of some anti-V2 MAbs – binding enhanced by some anti-CD4 binding site MAbs [Moore & Sodroski(1996)] • 110.I: Epitope suggested to be RAFVTIGK – V3 MAbs 9284, BAT123, 110.5, and 110.1 could each significantly increase gp120 dissociation from virus, mimicking sCD4, and expose the gp41 epitope for MAb 50-69, in contrast to anti-V2 MAbs [Poignard et al.(1996a)] 					
341 110.J	gp120(V3 loop)	gp120	?	?	?	(unk)
Donor:	F. Traincard, Pasteur Institute, France					
References:	[Thali et al.(1993), Moore & Sodroski(1996)]					
NOTES:						
	<ul style="list-style-type: none"> • 110.J: Inhibits sCD4-inducible anti-CD4 binding site MAb 48d [Thali et al.(1993)] • 110.J: Binds to carboxy-terminal side of the V3 loop – reciprocal binding inhibition with other anti-V3 and anti-C4 MAbs – and reciprocal enhanced binding of some anti-V2 MAbs and anti-CD4 binding site MAbs [Moore & Sodroski(1996)] 					
342 G3-1472	gp120(V3 loop)	gp120	?	?	?	(unk)
Donor:	M. Fung					
References:	[Moore & Sodroski(1996)]					
NOTES:						
	<ul style="list-style-type: none"> • G3-1472: Binds to carboxy-terminal side of the V3 loop – reciprocal binding inhibition with other anti-V3 and anti-C4 MAbs – reciprocal enhanced binding of some anti-V2 MAbs and anti-CD4 binding site MAbs [Moore & Sodroski(1996)] 					
343 AG1121	gp120(V3 loop)	gp120	?	L	?	(unk)
Donor:	AGMED, Inc, Bedford MA, commercial					
References:	[Sullivan et al.(1995)]					
NOTES:						
	<ul style="list-style-type: none"> • AG1121: Recognizes monomeric gp120 from T-cell adapted line HXBc2 and primary isolate 89.6 equally well, but 89.6 was three-fold less sensitive to neutralization by AG1121 than HXBc2 [Sullivan et al.(1995)] 					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
344 110.6	gp120(V3 BRU)	gp120(313-320)	RGPGR ^A AFV	L (weak)	BRU infected cell lysates	murine(IgG _{1λ})
	References: [Kinney Thomas et al.(1988), Pirofski et al.(1993), Langedijk et al.(1992)]					
	NOTES:					
	• 110.6: Variable region sequenced – heavy chain: V J558-146b.1 α , D closest to DSP16.2, J _H 3 – light chain: V λ 1, J λ 1 [Pirofski et al.(1993)]					
345 BAT123	gp120(V3 308-324)	gp120(308-324)	RIRIQRG ^B GR-AFVTIGK	L	Inact IIIB	murine(IgG _{1κ})
	Donor: Tanox Biosystems Inc and David Ho, ADARC, NY					
	References: [Fung et al.(1987), Liou et al.(1989), Fung et al.(1990), Moore & Ho(1993), Safrit et al.(1993), Thali et al.(1993), Pirofski et al.(1993), Gauduin et al.(1995), Sattentau & Moore(1995), Poignard et al.(1996a)]					
	NOTES:					
	• BAT123: Anti-idiotypic MAB, AB19-4i, stimulates anti-anti-ID which neutralizes MN and IIIB [Fung et al.(1990)]					
	• BAT123: Called BAT-123 – conformational, does not bind well to denatured gp120 – not reactive with SF-2 gp120 – does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)]					
	• BAT123: Passive transfer to Hu-PBS-SCID mice confers protection against challenge with homologous cell-free virus [Safrit et al.(1993)]					
	• BAT123: Variable region sequenced – heavy chain: V 3660-SB32, D unknown, J _H 3 – light chain: V κ 21, J κ 2 [Pirofski et al.(1993)]					
	• BAT123: Passive transfer of BAT123 to hu-PBL-SCID mice 1 hour prior to inoculation with HIV-1 LAI, or up to four hours post-exposure could protect mice from infection – the protection, like the MAB, was specific for the viral strain LAI [Gauduin et al.(1995)]					
	• BAT123: Binds with high affinity to monomer and oligomer, rapid association and potent neutralization of lab strain [Sattentau & Moore(1995)]					
	• BAT123: Epitope described as RGPGR ^A AFV ^B TIGK – V3 MAbs 9284, BAT123, 110.5, and 110.I could each significantly increase gp120 dissociation from virus (BAT123 less so than the others), mimicking sCD4, and expose the gp41 epitope for MAb 50-69, in contrast to anti-V2 MAbs [Poignard et al.(1996a)]					
346 CGP 47 439	gp120(V3 tip)	gp120(308-324)	?	L	III ^C gp120	BAT123-human Ig chimera
	References: [Liou et al.(1989), Safrit et al.(1993), Gunthard et al.(1994)]					
	NOTES:					
	• CGP 47 439: passive transfer to Hu-PBS-SCID mice confers protection against challenge with homologous cell-free virus – BAT123-human Ig chimera [Safrit et al.(1993)]					
	• CGP 47 439: PhaseI/IIA clinical trial studying multidosage tolerability, immunogenicity and pharmacokinetic responses – GP 47 439 was well tolerated, serum t _{1/2} was 8-16 days, and a virus burden reduction was noted in some patients [Gunthard et al.(1994)]					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
347 10F10	gp120(V3 MN)	gp120(308-322)	RKRHIGPGRAYTT	L	Peptide	murine(IgG ₁)
	References:	[Duarte et al.(1994)]				
NOTES:	<ul style="list-style-type: none"> • 10F10: Putative epitope lies within IHIGPGRAYTT – generated by multi-epitope polypeptide immunization – recognize MN and SC (TRSIHIGPGRAYTT) peptides, lower affinity for SF2 [Duarte et al.(1994)] 					
348 2C4	gp120(V3 MN)	gp120(308-322)	RKRHIGPGRAYTT	L (MN)	Peptide	murine(IgG _{2a})
	References:	[Duarte et al.(1994)]				
NOTES:	<ul style="list-style-type: none"> • 2C4: Putative epitope lies within IHIGPGRAYTT – neutralizes MN, not IIIB and SF2 – generated by multi-epitope polypeptide immunization – recognize MN and SC (TRSIHIGPGRAYTT) peptides, lower affinity for SF2 [Duarte et al.(1994)] 					
349 19b	gp120(V3)	gp120(310-322)	-I—G-FY-T	L	HIV-1 infection	human(IgG)
	Donor:	James Robinson, Tulane University, LA				
	References:	[Scott Jr et al.(1990), Moore et al.(1994b), Moore et al.(1994a), Sattentau(1995), Moore et al.(1995b), Moore et al.(1995a), Moore & Ho(1995), Gauduin et al.(1996), Wu et al.(1996), Trkola et al.(1996a), D'Souza et al.(1997), Binley et al.(1997), Fouts et al.(1997), Ugolini et al.(1997)]				
NOTES:	<ul style="list-style-type: none"> • 19b: V3 loop binding MAbs that is more broadly clade cross-reactive than most (binds to 19/29 clade B and 10/12 clade E gp120s) [Moore et al.(1994b)] • 19b: Competition studies with human sera from seroconverting individuals showed that anti-CD4 BS antibodies can arise very early in infection, comparable or prior to anti-V3 antibodies [Moore et al.(1994a)] • 19b: Formalin inactivation of virus at 0.1% formalin for 10 hours at 4 degrees was optimal for inactivation of virus while maintaining epitope integrity [Sattentau et al.(1995)] • 19b: Binds to some gp120s from clades A,B,C,E, and F – weakly neutralized some B and one C clade virus [Moore et al.(1995b)] • 19b: Despite broad gp120 binding reactivity, not broadly neutralizing [Moore et al.(1995a)] • 19b: Review: more broadly cross-reactive than anti-V3 tip MAb 447-D [Moore & Ho(1995)] • 19b: Not as effective as IgG1b12 at neutralization <i>ex vivo</i> of virus direct from plasma of HIV-1 infected individuals [Gauduin et al.(1996)] • 19b: MIP-1α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4 – binding of 19b blocks this inhibition [Wu et al.(1996)] • 19b: Inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)] • 19b: In a multilaboratory blinded study, failed to consistently neutralize any of nine B clade primary isolates – there were four sequences with variations the defined epitope among the 9 isolates tested [D'Souza et al.(1997)] • 19b: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)] • 19b: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 19b bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)] • 19b: Viral binding inhibition by 19b was weakly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]^c 					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
350 G3-523	gp120(V3 308-322)	gp120(310-324)	RIGRGPGRAFVTIGK	?		murine(unk)
	References: [Matsushita et al.(1988), Jagodzinski et al.(1996)]					
NOTES:	<ul style="list-style-type: none"> • G3-523 : The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus - CRDS inhibits G3-523 binding [Jagodzinski et al.(1996)] 					
351 10/54	gp120(V3 311-321 HXB10)	gp120(313-323)	RGPGRAFVTIG	L (HXB10)	rgp120 BH10	rat (IgG ₁)
	References: [McKeating et al.(1992a), McKeating et al.(1993a), McKeating et al.(1993b)]					
NOTES:	<ul style="list-style-type: none"> • 10/54: Binding to virion gp120 enhanced by sCD4 [McKeating et al.(1992a)] • 10/54: Studied in the context of a neutralization escape mutant [McKeating et al.(1993a)] 					
352 10/36e	gp120(V3 311-321 HXB10)	gp120(313-323)	RGPGRAFVTIG	L (HXB10)	rgp120 BH10	rat (IgG _{2a})
	References: [McKeating et al.(1992a), McKeating et al.(1993b)]					
NOTES:	<ul style="list-style-type: none"> • 10/36e: Binding to virion gp120 enhanced by sCD4 [McKeating et al.(1992a)] 					
353 11/85b	gp120(V3 311-321 HXB10)	gp120(313-323)	RGPGRAFVTIG	L (HXB2)	rgp120 BH10	rat (IgG _{2b})
	References: [McKeating et al.(1992a), McKeating et al.(1993b)]					
NOTES:	<ul style="list-style-type: none"> • 11/85b: Binding to virion gp120 enhanced by sCD4 [McKeating et al.(1992a)] 					
354 loop 2	gp120(V3)	gp120(311-322)	SISGPGRAFYTG	L	HIV-1 infection	human Fab(unk)
	References: [Barbas III et al.(1993), Moore et al.(1994b), Wu et al.(1996), Ditzel et al.(1997), Ugolini et al.(1997)]					
NOTES:	<ul style="list-style-type: none"> • loop 2: Sequences of the heavy and light chain Fab variable regions were generated [Barbas III et al.(1993)] • loop 2: Called Loop 2 – shows modest cross-reactivity among B clade gp120s, little outside B clade [Moore et al.(1994b)] • loop 2: MIP-1α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4 – binding of loop 2 blocks this inhibition • loop 2: Binds to gp120 from MN and SF2 but not LAI [Ditzel et al.(1997)] • loop 2: Viral binding inhibition by loop 2 Mab or Fab was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)] 					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
355 41.1	gp120(V3 HXB10)	gp120	conformation dependent	L (HXB2)	rgp120 BH10	rat(IgG _{2a})

Donor: J. Cordell, Institute for Cancer Research, Sutton, Surrey, UK

References: [McKeating et al.(1992a), McKeating et al.(1993b), Klasse et al.(1993a), McLain & Dommock(1994), Armstrong & Dommock(1996), Armstrong et al.(1996), Jeffs et al.(1996), Ugolini et al.(1997)]

NOTES:

- 41.1: Also called ICR41.1i and ICR41
- 41.1: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to conformationally sensitive neutralizing MAbs – neutralization efficiency of 41.1 is not affected [Reitz Jr. et al.(1988), Klasse et al.(1993a)]
- 41.1: Called ICR41.1i – Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively – neutralization mediated by 3 molecules of IgG per virion – most efficient at neutralization of the three MAbs studied – acts with multi-hit kinetics [McLain & Dommock(1994)]
- 41.1: Called ICR41.1i – IgG_{2c}? – Neutralization was affected if the Ab was added after the virus bound to the host cells at 24 degrees C or below [Armstrong & Dommock(1996)]
- 41.1: Called ICR41.1i – Neutralization occurs by blocking a post-fusion internalization event, in contrast to MAb F58 [Armstrong et al.(1996)]
- 41.1: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs et al.(1996)]
- 41.1: Viral binding inhibition by 41.1 was weakly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]

356 DO142-10	gp120(V3 MN)	gp120()	SISCPGGRAFYTG	L	HIV-1 infection	human Fab(IgG ₁)
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References: [Ditzel et al.(1997)]

NOTES:

- DO142-10: Phage expression libraries panned against MN peptide were used to select Fab DO142-10 – Fab binds MN gp120, but not a primary isolate rec gp120 [Ditzel et al.(1997)]

HIV Monoclonal Antibodies

Mab ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
357 257-D	gp120(V3 MN)	gp120(309-313)	KRIHI	L	HIV-1 infection	human(IgG _{1,λ})

Donor: Susan Zolla-Pazner (NYU Med. Center)

References: [Gorny et al.(1991), D'Souza et al.(1991), Karwowska et al.(1992b), Gorny et al.(1993), Cavacini et al.(1993a), Spear et al.(1993), D'Souza et al.(1994), VanCott et al.(1994), D'Souza et al.(1995), Zolla-Pazner et al.(1995), Schutten et al.(1995a), Schutten et al.(1995b), Fontenot et al.(1995), Wisniewski et al.(1996), Schutten et al.(1996), Schutten et al.(1997), Stamatas et al.(1997)]

NOTES:

- 257-D: Also called 257-2-D-IV and 257-D-IV
- 257-D: Called 257-2-D-IV – potent neutralizing MAb [D'Souza et al.(1991)]
- 257-D: Reacts with MN, NY5, CDC4 and SF2, does not cross-react with RF, WM52, or HXB2 [Karwowska et al.(1992b)]
- 257-D: Neutralizes MN – binds SF2: KSIY1 – specificity: MN, SF2, NY5, RF. [Gorny et al.(1993)]
- 257-D: Additive MN or SF2 neutralization when combined with CD4 binding site MAb F105 – does not neutralize RF [Cavacini et al.(1993a)]
- 257-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG – complement mediated virolysis of MN, but not in the presence of sCD4 [Spear et al.(1993)]
- 257-D: Included a multi-lab study for antibody characterization and assay comparison – best NAb against MN, but not IIIB [D'Souza et al.(1994)]
- 257-D: Potent MN neutralization, slow dissociation constant [VanCott et al.(1994)]
- 257-D: Called 257-2-D-IV – could neutralize MN and closely related JRCSF, but not 2 B subtype and 1 D subtype primary isolates in a multi-laboratory study involving 11 labs [D'Souza et al.(1995)]
- 257-D: In serotyping study using flow-cytometry, bound only to virus with KRIHI [Zolla-Pazner et al.(1995)]
- 257-D: Only inhibition of SI phenotype virus, and strong enhancement of NSI phenotype chimeric viruses, that incorporated different envs from the same donor [Schutten et al.(1995a)]
- 257-D: Comparable affinity for SI and NSI viruses, in contrast to MAb MN215 [Schutten et al.(1995b)]
- 257-D: 257-D is V_H5 – V-region heavy chain usage was examined and a bias of enhanced V_H1 and V_H4, and reduced V_H3, was noted among HIV infected individuals [Wisniewski et al.(1996)]
- 257-D: IIIB neutralizing Mabs *in vitro* fail to neutralize in a mouse model *in vivo* [Schutten et al.(1996)]
- 257-D: Neutralized (>90%) an SI-env chimeric virus and enhanced (>200%) an NSI-env chimeric virus [Schutten et al.(1997)]
- 257-D: Binds less extensively than MAb 391-95D on the surface of HIV-1 isolates SF162 and SF128A – neutralizes less potently than 391-95D – stronger neutralization of primary macrophage targets than PBMNC [Stamatas et al.(1997)]
- 257-D: UK Medical Research Council AIDS reagent: ARP3023
- 257-D: NIH AIDS Research and Reference Reagent Program: 1510

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
358 4117C	gp120(V3)	gp120(311-317)	IIXIGPGR	L	HIV-1 infection	human(IgG _{1,λ})
References:	[Tilley et al.(1991a), Tilley et al.(1992), di Marzo Veronese et al.(1993), Pinter et al.(1993a), Pinter et al.(1993b)]					
NOTES:						
• 4117C: Potent neutralizing activity against MN, SF-2, and NY-5 – synergy with CD4BS MAb 1125H [Tilley et al.(1991a)]						
• 4117C: Neutralizes SF2 and MN synergistically combined with anti-CD4 binding site discontinuous MAbs [Pinter et al.(1993a), Tilley et al.(1992)]						
• 4117C: Binds V3 loop – does not immunoprecipitate soluble gp120, does react with gp120 on intact virions [Pinter et al.(1993b)]						
359 41148D	gp120(V3 MN)	gp120(309-315)	KRIIHGP	L	HIV-1 infection	human(IgG)
References:	[Pinter et al.(1993b)]					
NOTES:						
• 41148D: Neutralizes less potently than 4117C, reacts with MN, IIIB, SF2 [Pinter et al.(1993b)]						
360 453-D	gp120(V3 MN)	gp120(311-317)	IHIIGPGR	L	HIV-1 infection	human(IgG _{1,λ})
References:	[Gorny et al.(1993), VanCott et al.(1994), Fontenot et al.(1995)]					
NOTES:						
• 453-D: Neutralizes MN – binds SF2: IYIGPGR – specificity: MN, SF2, NY5, RF [Gorny et al.(1993)]						
• 453-D: Moderate homologous neutralization, moderately slow dissociation rate [VanCott et al.(1994)]						
• 453-D: 453-D : Called 453, epitope described as KRIIHGPGR – the tip of the V3 loop was presented in a mucin backbone – higher valency correlates with stronger affinity constant [Fontenot et al.(1995)]						
361 504-D	gp120(V3)	gp120(311-317)	IHIIGPGR	L	HIV-1 infection	human(IgG _{1,κ})
References:	[Gorny et al.(1993)]					
NOTES:						
• 504-D: 504-D – Neutralizes MN – binds SF2: IYIGPGR [Gorny et al.(1993)]						
362 418-D	gp120(V3)	gp120(312-318)	HIGPGRA	L	HIV-1 infection	human(IgG _{1,κ})
References:	[Karwowska et al.(1992b), Gorny et al.(1993)]					
NOTES:						
• 418-D: MN strain specific, does not cross-react with SF2, NY5, RF, CDC4 WM52 or HXB2 [Karwowska et al.(1992b)]						
• 418-D: Neutralizes MN, does not bind to SF2 or HXB2 [Gorny et al.(1993)]						

HIV Monoclonal Antibodies

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
363 311-11-D	gp120(V3)	gp120(309-315)	KRIHIGP	L	HIV-1 infection	human(IgG _{1,λ})
References: [Gorny et al.(1993), Spear et al.(1993)]						
NOTES:						
• 311-11-D: Neutralizes MN – binds SF2: KSIYIGP [Gorny et al.(1993)]						
• 311-11-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG [Spear et al.(1993)]						
364 391/95-D	gp120(V3)	gp120(308-322)	RKRHIIGPGRAFYTT	L	HIV-1 infection	human(IgG _{1,κ})
References: [Gorny et al.(1993), Fontenot et al.(1995), Seligman et al.(1996), Stamatatos et al.(1997)]						
NOTES:						
• 391/95-D: Also called 391-95D						
• 391/95-D: Neutralizes MN – binds to SF2, not IIIB [Gorny et al.(1993)]						
• 391/95-D: Competition ELISAs with serial deletions estimated the epitope to be KRIHIGPGRAFY – unconstrained peptide had higher affinity than cyclic [Seligman et al.(1996)]						
• 391/95-D: Called 391-95D – binds more extensively than MAb 257-D on the surface of HIV-1 isolates SF162 and SF128A – neutralizes more potently than 257-D – stronger neutralization of primary macrophage targets than PBM – binding post-gp120-sCD4 association is related to anti-V3 Abs neutralizing capacity [Stamatatos et al.(1997)]						
365 412-D	gp120(V3 MN)	gp120(308-322)	RKRHIIGPGRAFYTT	L	HIV-1 infection	human(IgG _{1,κ})
References: [Gorny et al.(1993), Spear et al.(1993), VanCott et al.(1994), Fontenot et al.(1995)]						
NOTES:						
• 412-D: Neutralizes MN, does not bind SF2 or HXB2 – not reactive with hexa or heptapeptides by PEPscan						
• 412-D: [Gorny et al.(1993)]						
• 412-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG [Spear et al.(1993)]						
• 412-D: Relatively rapid dissociation and weak homologous neutralization – also called 412-10D [VanCott et al.(1994)]						
• 412-D: Called 412 – The tip of the V3 loop was presented in a mucin backbone – higher valency correlates with stronger affinity constant [Fontenot et al.(1995)]						
366 MN215	gp120(V3 MN)	gp120(310-324)	RIHIGPGRAFYTTKN		HIV-1 infection	human(IgG ₁)
References: [Schutten et al.(1995b)]						
NOTES:						
• MN215: Minimum epitope for MAB using the Dutch consensus is AFYTTGE, different than defined for MN – generated by EBV transformation of PBM – displayed higher affinity for NSI than for SI glycoproteins – amino acids HIGP were essential for binding [Schutten et al.(1995b)]						

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
367 SP.BAL114	gp120(V3 BAL)	gp120(310-319)	SIHIGPGRRAF	L	?	murine?(IgG _{2a} κ)
	References: [Arendrup et al.(1995)]					
NOTES:						
	• SP.BAL114: Authors suggest that during in vivo immunoselection of escape virus, the V3 domain gains increasing resemblance to that of lab strains [Arendrup et al.(1995)]					
368 SP.SF2:104	gp120(V3 SF2)	gp120(310-319)	SIYIGPGRRAF	L	?	murine?(IgG _{2a} κ)
	References: [Arendrup et al.(1995)]					
NOTES:						
	• SP.SF2:104: Authors suggest that during in vivo immunoselection of escape virus, the V3 domain gains increasing resemblance to lab strains [Arendrup et al.(1995)]					
369 477-D	gp120(V3)	gp120(312-315)	HIGP	L	HIV-1 infection	human(IgG ₁ κ)
	References: [Gorny et al.(1993)]					
NOTES:						
	• 477-D: MN and SF2 strain specific, does not cross-react with NY5, RF, CDC4, WM52 or HXB2 [Karwowska et al.(1992b)]					
	• 477-D: Neutralizes MN – binds SF2: YIGP [Gorny et al.(1993)]					
370 μ5.5	gp120(V3 311-324 MN)	gp120(310-323)	RIHIGPGRAYTTG	L	?	murine(unk)
	Donor: T. Hattori, Kyoto U., Japan, and H. Schuitemaker and H. Huisman, Netherlands Red Cross					
NOTES:						
	• μ5.5: μ5.5: Binds MN but not IIIB infected HUT 78 cells, and blocks sCD4-induced 0.5β binding to MN [Maeda et al.(1992), D'Souza et al.(1994)]					
	• μ5.5: μ5.5: Included in a panel of antibodies used in a multi-lab study for antibody characterization, and binding and neutralization assay comparison [D'Souza et al.(1994)]					

HIV Monoclonal Antibodies

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
371 83.1	gp120(V3 312-318 MN)	gp120(311-317)	IXIGPGR	L	MN V3 Peptide	murine(IgG1)

References: [White-Scharf et al.(1993), Potts et al.(1993), Robert-Guroff et al.(1994), D'Souza et al.(1994), Moore et al.(1994b), Trkola et al.(1996a), Seligman et al.(1996), Binley et al.(1997)]

NOTES:

- 83.1: Epitope defined by peptide reactivity and changes in binding affinity with substitutions [White-Scharf et al.(1993)]
- 83.1: No synergistic neutralization of MN when combined with CD4BS MAb F105, some with sCD4 – synergistic neutralization with sCD4 of a field isolate [Potts et al.(1993)]
- 83.1: MN V3 loop in a HXB2 background allows enhanced FACS labeling of infected H9 cells and increased Ab affinity [Robert-Guroff et al.(1994)]
- 83.1: Included in a multi-lab study for antibody characterization and binding and neutralization assay comparison [D'Souza et al.(1994)]
- 83.1: Shows modest cross-reactivity among B clade gp120s, little outside B clade [Moore et al.(1994b)]
- 83.1: Inhibits gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola et al.(1996a)]
- 83.1: Competition ELISAs with serial deletions produced slightly longer estimate of epitope length than alanine substitution, RHIGPGR – unconstrained peptide had higher affinity than cyclic [Seligman et al.(1996)]
- 83.1: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)]

MAb ID	Location	WEAU	Sequence	Neutralizing	Immuno	Species(Isootype)
372 F58/H3	gp120(V3 307-316 IIIB)	gp120(310-319)	RIGRGPGRRAF	L	IIIB gp120	murine(IgG1 κ)

Donor: Dr. B. Wahren

References: [Akerblom et al.(1990), Brolden et al.(1990), Marks et al.(1992), Brolden et al.(1992), Arendrup et al.(1993), D'Souza et al.(1994), Duarte et al.(1994), Thali et al.(1994), Hinkula et al.(1994), Bolmstedt et al.(1996), Schutten et al.(1996), Pincus et al.(1996), Lundin et al.(1996), Armstrong et al.(1996)]

NOTES:

- F58/H3: F58/H3 is also called F58H3 and F58
- F58/H3: Neutralized multiple primary isolates with varying potency, no ADCC activity [Akerblom et al.(1990)]
- F58/H3: Variable domain sequenced and is identical to P4/D10 [Marks et al.(1992)]
- F58/H3: Neutralizes IIIB equally well when added before or after virus adhesion to target cells [Arendrup et al.(1993)]
- F58/H3: Included in a multi-lab study for antibody characterization and neutralization assay comparison [D'Souza et al.(1994)]
- F58/H3: Neutralizes IIIB but not SF2 or MN [Duarte et al.(1994)]
- F58/H3: gp41 mutation that confers resistance to neutralization by anti-CD4 binding site antibodies does not reduce neutralizing efficiency of this V3 region MAb [Thali et al.(1994)]
- F58/H3: Used for passive immunotherapy in seven late-stage HIV-infected patients – in 5/7 the serum level of p24 decreased [Hinkula et al.(1994)]
- F58/H3: Sera was obtained from guinea pigs vaccinated either with gp160, or with gp160 lacking N-linked glycans at N406, N448, and N463 – these sera could block equally well both the CD4 BS MAb GP13 and the V3 MAb F58/H3 [Bolmstedt et al.(1996)]
- F58/H3: IIIB neutralizing Mabs *in vitro* fail to neutralize in a mouse model *in vivo* [Schutten et al.(1996)]
- F58/H3: A panel immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus et al.(1996)]
- F58/H3: MAb F58/H3 was used to bind peptides from random libraries – the binding motif in V3 is -I-GPGRA-, but peptides -GPGRA-, -FRILG-, and -WR(M/A)LG- were selected – the -GPGRA- peptide presented on phage was used as an immunogen in rabbits, and resulting polyclonal sera neutralized HIV-1 SF2 [Lundin et al.(1996)]
- F58/H3: Called F58 – Neutralization occurs by inhibiting viral fusion with the cell and internalization of the viral core, in contrast to MAb 41.1 [Armstrong et al.(1996)]

HIV Monoclonal Antibodies

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
373 SP.SF2:104	gp120(V3 SF2)	gp120(310-319)	SIYIGPGRAY			(IgG _{2aκ})
	References: [Arendrup et al.(1993)]					
NOTES:						
	• SP.SF2:104: Anti-V3 antibody that could neutralize primary virus isolated from a time point when neutralization resistance of autologous virus [Arendrup et al.(1993)]					
374 A47/B1	gp120(V3 307-316 IIIB)	gp120(311-320)	IQRGPGRAYV	L	IIIB gp120	murine(IgG)
	References: [Akerblom et al.(1990)]					
375 G44/H7	gp120(V3 307-316 IIIB)	gp120(311-320)	IQRGPGRAYV	L	IIIB gp120	murine(IgG)
	References: [Akerblom et al.(1990)]					
376 D59/A2	gp120(V3 307-316 IIIB)	gp120(311-320)	IQRGPGRAYV	L	IIIB gp120	murine(IgG)
	References: [Akerblom et al.(1990)]					
377 IIIB-34 V3	gp120(V3 308-316 IIIB)	gp120(311-319)	IQRGPGRAY	L	Peptide	murine(IgG ₁)
	References: [Laman et al.(1992), Laman et al.(1993)]					
NOTES:						
	• IIIB-34 V3: Neutralizes IIIB but not MN – QXGPG are critical amino acids for binding by pepscan analysis [Laman et al.(1992)]					
	• IIIB-34 V3: Called IIIB-V3-34 – IIIB strain specific neutralization – binding is reduced somewhat by DTT or SDS-DTT, enhanced by NP40, but binds to native and denatured gp120 [Laman et al.(1993)]					
	• IIIB-34 V3: UK Medical Research Council AIDS reagent: ARP3047					
378 IIIB-13 V3	gp120(V3 308-316 IIIB)	gp120(311-319)	IQRGPGRAY	L	Peptide	murine(IgG ₁)
	References: [Laman et al.(1992), Laman et al.(1993), D'Souza et al.(1994), Watkins et al.(1993)]					
NOTES:						
	• IIIB-13 V3: Also known as 1044-13 and as IIIB-V3-13 (J. P. Moore, per. comm.)					
	• IIIB-13 V3: Neutralizes IIIB but not MN [Laman et al.(1992)]					
	• IIIB-13 V3: Included in a panel of antibodies used in a multi-lab study for antibody characterization and assay comparison, some neutralization of strains other than IIIB [D'Souza et al.(1994)]					
	• IIIB-13 V3: Called IIIB-V3-13 – a neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – IIIB-V3-13 neutralization was only slightly reduced by this mutation [Watkins et al.(1993)]					
	• IIIB-13 V3: UK Medical Research Council AIDS reagent: ARP3046					
	• IIIB-13 V3: NIH AIDS Research and Reference Reagent Program: 1727					

III-90
DEC 97

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
379 M77	gp120(V3 IIIB)	gp120(309-322)	I ₁ I ₂ QRGPGRAFV ₁ I	L	HIV-1 infection	human(IgG)

Donor: Advanced BioScience Laboratories, Rockville, MD, commercial

References: [Pal et al.(1992), di Marzo Veronese et al.(1992), di Marzo Veronese et al.(1993), Watkins et al.(1993), Cook et al.(1994), Devico et al.(1995), Denisova et al.(1995), Denisova et al.(1995), Watkins et al.(1996)]

NOTES:

- M77: IIIB-specific MAb, immunoprecipitates deglycosylated form [di Marzo Veronese et al.(1992)]
- M77: Antibody binding to viral isolates from IIIB infected lab worker followed through time – A to T substitution resulted in the loss of neutralization and native gp120 binding, but not peptide binding [di Marzo Veronese et al.(1993)]
- M77: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer *in vitro* [Cook et al.(1994)]
- M77: Reacted with both reduced and non-reduced covalently cross-linked gp120-CD4 complex [Devico et al.(1995)]
- M77: Conformational rearrangements upon binding of M77 to gp120 generates novel epitopes called metatopes [Denisova et al.(1995)]
- M77: Stated to be a murine MAb – a neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – M77 neutralization was only slightly reduced by this mutation [Watkins et al.(1993)]
- M77: Used M77 bound to gp120 as an immunogen – analysis of polyclonal and monoclonal (62 MAbs were generated) response suggests the M77-gp120 immunogen generated MAbs to more linear epitopes than gp120 alone or gp120 bound to CD4 [Denisova et al. (1996)]
- M77: Native M77 is highly strain specific, and V3 binding is primarily dependent on its heavy chain – a light chain switched Fab version of M77 could recognize HIV-1 strains that had substitutions on the left side of the V3 loop – R in GPGR is likely to be critical for binding [Watkins et al.(1996)]

HIV Monoclonal Antibodies

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
380 268-D	gp120(V3 MN)	gp120(312-317)	HIGPGR	L	HIV-1 infection	human(IgG1 λ)
	Donor: Susan Zolla-Pazner (NYU Med. Center)					
	References: [Gorny et al.(1991), D'Souza et al.(1991), Karwowska et al.(1992b), Gorny et al.(1993), Spear et al.(1993), VanCott et al.(1994), Zolla-Pazner et al.(1995), Fontenot et al.(1995), McKeating et al.(1996), Wisniewski et al.(1996), Stamatas et al.(1997)]					
	NOTES:					
	<ul style="list-style-type: none"> • 268-D: Also called 268-11-D-IV and 268D • 268-D: Called 268-11-D-IV – strain specific weakly neutralizing [D'Souza et al.(1991)] • 268-D: Reacts with MN, NY5, CDC4, RF and SF2, does not cross-react with WM52 or HXB2 [Karwowska et al.(1992b)] • 268-D: Neutralizes MN – binds SF2: YIGPGR – specificity: MN, SF2, NY5, RF, CDC4 [Gorry et al.(1993)] • 268-D: Mediated deposition of complement component C3 on HIV infected cells, but not in the presence of sCD4 [Spear et al.(1993)] • 268-D: Moderate dissociation rate and homologous neutralization titer [VanCott et al.(1994)] • 268-D: Serotyping study using flow-cytometry, if H of HIGPGR was substituted in virus, 268-D did not bind [Zolla-Pazner et al.(1995)] • 268-D: Failed to neutralize HXB2 and chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)] • 268-D: 268-D is V_H4 – V-region heavy chain usage was examined and a bias of enhanced V_H1 and V_H4, and reduced V_H3, was noted among HIV infected individuals [Wisniewski et al.(1996)] • 268-D: Poor reactivity against HIV-1 isolates SF162 and SF128A and no neutralization, in contrast to MAbs 391/95-D and 257-D [Stamatas et al.(1997)] • 268-D: UK Medical Research Council AIDS reagent: AIP3024 • 268-D: NIH AIDS Research and Reference Reagent Program: 1511 					
381 polyclonal	gp120(V3 MN)	gp120(313-320)	IGPGRAFY	L	gp120- <i>B. abortus</i> complex (SF2 or MN)	murine(IgG2a)
	References: [Golding et al.(1995)]					
	NOTES:					
	<ul style="list-style-type: none"> • polyclonal: Ab is evoked even in mice depleted of CD4+ cells 					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunoigen	Species(Isotype)
382	0.5 β gp120(V3 316-330 HXB2)	gp120(313-326)	RGPGRGAFVTIGKIG	L (IIIB)	IIIB Env	murine(IgG1 κ)

Donor: Shuzo Matsushita or Toshio Hattori of Kumamoto University

References: [Matsushita et al.(1988), Skinner et al.(1988b), Skinner et al.(1990), D'Souza et al.(1991), Matsushita et al.(1992), Nara et al.(1990), McKeaning et al.(1992a), Sperlich et al.(1993), di Marzo Veronese et al.(1993), Moore et al.(1993b), Klasse et al.(1993a), Watkins et al.(1993), Cook et al.(1994), Thali et al.(1994), Okada et al.(1994), Bouclet et al.(1994), Broder et al.(1994), Zvi et al.(1995b), Zvi et al.(1995a), Jagodzinski et al.(1996), Warrier et al.(1996), McDougal et al.(1996), Jeffs et al.(1996), Huang et al.(1997), Zvi et al.(1997)]

NOTES:

- 0.5 β : Also called 0.5 beta
- 0.5 β ; 0.5 β : Type-specific neutralization of IIIB – does not neutralize MN or RF [Matsushita et al.(1988), Skinner et al.(1988b)]
- 0.5 β ; 0.5 β : Emergence of virus resistant to MAb 0.5 β and autologous sera neutralization in IIIB infected chimps [Nara et al.(1990)]
- 0.5 β ; 0.5 β : Potent neutralizing activity [D'Souza et al.(1991)]
- 0.5 β ; 0.5 β : Chimeric mouse-human MAb C β 1 was constructed by combining the human C γ 1 and C κ constant regions with the 0.5 β murine MAb – ADCC and neutralizing activity[Matsushita et al.(1992)]
- 0.5 β ; 0.5 β : sCD4 causes loss of IIIB type-specificity, allowing binding and neutralization of MN, in contrast to MAb μ 5.5 [Maeda et al.(1992)]
- 0.5 β ; 0.5 β : Monoclonal anti-idiotype antibodies that mimic the 0.5 β epitope were generated [Sperlich et al.(1993)]
- 0.5 β ; 0.5 β : Neutralization of virus carrying a A to T substitution (contrast with MAb M77) [di Marzo Veronese et al.(1993)]
- 0.5 β ; 0.5 β : Binding to native gp120 100-300 fold greater than to denatured [Moore et al.(1993b)]
- 0.5 β ; 0.5 β : The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to some antiserum and conformationally sensitive neutralizing MAbs – neutralization efficiency of 0.5 β is not affected [Reitz Jr. et al.(1988), Klasse et al.(1993a)]
- 0.5 β ; 0.5 β : A neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – of the MAbs tested , 0.5 β neutralization was the most profoundly affected by this mutation [Watkins et al.(1993)]
- 0.5 β ; 0.5 β : MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer *in vitro* [Cook et al.(1994)]
- 0.5 β ; 0.5 β : gp41 mutation that confers resistance to neutralization by anti-CD4 binding site antibodies does not reduce neutralizing efficiency of this V3 region MAb [Thali et al.(1994)]

HIV Monoclonal Antibodies

MAb ID	Description	Properties and References
382	0.5 β (cont.)	<ul style="list-style-type: none">• 0.5β: Binding domain aa 310-319: RGGGRAFVTIGKIG – mutations in the V3 loop from basic residues can destroy virus infectivity and syncytium formation: 306 R/T,309 R/T and 313 R/G can also reduce binding of V3 MAbs with two different binding sites: 9284 and 0.5β [Okada et al.(1994)]• 0.5β: Type-specific neutralization of IIIB – does not neutralize SF2 [Broder et al.(1994)]• 0.5β: The interactions of the peptide RKSIRIQRGPGRAFVLT 0.5β were studied by NMR, and hydrophobic interactions between the two IIs and the V form the base of a 12 amino acid loop with GPGR at the apex[Zvi et al.(1995b)]• 0.5β: NMR of 0.5β bound NNTRKSIRIQRGPGRAFVLT suggests that the bound amino acids are in the region SIRIQRGPGRAFVLT [Zvi et al.(1995a)]• 0.5β: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – CRDS inhibits 0.5β binding – 0.5β epitope described as GPGRAFVTTG [Jagodzinski et al.(1996)]• 0.5β: Synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G [Warrier et al.(1996)]• 0.5β: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs et al.(1996)]• 0.5β: Relative to the native peptide, an O-linked α-galactosamine modified V3 peptide enhanced binding to 0.5β, while an N-linked β-glucosamine modified peptide showed reduced binding [Huang et al.(1997)]• 0.5β: 0.5β: The structure of a 17 amino acid V3 peptide bound to the FAb was studied using NMR [Zvi et al.(1997)]• 0.5β: 0.5β: UK Medical Research Council AIDS reagent: ARP3025• 0.5β: 0.5β: NIH AIDS Research and Reference Reagent Program: 1591

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(IsoType)
383 924	gp120(V3 309-318 IIIB)	gp120(308-316)	RKSIRIQRGPG	vaccinia-gp160 IIIB	murine(IgG _{1κ})	
	References: [Chesebro & Wehrly(1988), Pincus et al.(1991), Cook et al.(1994), Pincus et al.(1996)]					
	NOTES:					
	<ul style="list-style-type: none"> • 924: HIV IIIB strain specific [Chesebro & Wehrly(1988)] • 924: Epitope sequence is based on database count of a specified location – 924-RAC immunotoxin is IIIB strain-specific [Pincus et al.(1991)] • 924: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer <i>in vitro</i> [Cook et al.(1994)] • 924: A panel immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus et al.(1996)] 					
384 907	gp120(V3 309-318)	gp120(308-316)	RKSIRIQRGPG	L	vaccinia-gp160 IIIB	murine(IgG _{1κ})
	References: [Chesebro & Wehrly(1988), Pincus et al.(1989), Pincus et al.(1991), Pincus et al.(1996)]					
	NOTES:					
	<ul style="list-style-type: none"> • 907: Strain specific binding, and neutralization of only the LAV strain [Chesebro & Wehrly(1988)] • 907: Coupled to ricin A chain (RAC), MAB 907 inhibited protein synthesis and cell growth in HIV-infected cells [Pincus et al.(1989)] • 907: Epitope sequence is based on database count of a specified location – 924-RAC immunotoxin is IIIB strain-specific [Pincus et al.(1991)] • 907: A panel immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus et al.(1996)] 					
385 Cβ1	gp120(V3 316-330 HXB2)	gp120(313-326)	RGPGRAFVTIGKIG	L	IIIB Env	human (IgG ₁) 0.5β chimaera
	References: [Emini et al.(1992)]					
	NOTES:					
	<ul style="list-style-type: none"> • C_β1: C_β1: passive transfer to chimpanzees confers protection against challenge with homologous cell-free virus – mouse 0.5β human IgG₁ chimera [Emini et al.(1992)] 					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
386 386-D	gp120(V3 MN)	gp120(312-317)	HIGPGR	L	HIV-1 infection	human(IgG _{1,λ})
	References:	[Karwowska et al.(1992b), Gorny et al.(1993), VanCott et al.(1994), Fontenot et al.(1995)]				
NOTES:						
	• 386-D: Neutralizes MN – binds SF2: YIGPGR – specificity: MN, SF2, NY5, RF, CDC4 [Gorny et al.(1993)]					
	• 386-D: Slow dissociation rate, potent homologous neutralization [VanCott et al.(1994)]					
387 5021	gp120(V3)	gp120(312-318)	QrGPGRa	L	15 mer BH10 V3 peptide	murine(IgG)
	References:	[Durda et al.(1988), Durda et al.(1990), Langedijk et al.(1991), Moore et al.(1993b)]				
NOTES:						
	• 5021: Generation and fine mapping of murine MAbs [Langedijk et al.(1991)]					
	• 5021: Binding to native gp120 100-300 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore et al.(1993b)]					
388 5042	gp120(V3)	gp120(312-318)	QRGPGRA	L	peptide	murine(unk)
	References:	[Durda et al.(1988), Durda et al.(1990), Moore et al.(1993b)]				
NOTES:						
	• 5042: Binding to native gp120 100-300 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore et al.(1993b)]					
389 F58/D1	gp120(V3)	gp120(311-318)	IXXGPGR	L	virus derived gp120	human(unk)
	References:	[Akerblom et al.(1990), Brolden et al.(1991), Moore et al.(1993b)]				
NOTES:						
	• F58/D1: Binding to native gp120 1-3 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore et al.(1993b)]					
390 P1/D12	gp120(V3)	gp120(311-318)	IXXGPGR	L	virus derived IIIB gp120	murine(IgG)
	References:	[Akerblom et al.(1990), Moore et al.(1993b)]				
NOTES:						
	• P1/D12: Binding to native gp120 1-3 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore et al.(1993b)]					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
391 P4/D10	gp120(V3)	gp120(311-318)	IXXGPGRA	L	virus derived IIIB gp120	murine(IgG _{1,κ})
References:	[Akerblom et al.(1990), Brolden et al.(1990), Brolden et al.(1991), Marks et al.(1992), Moore et al.(1993b), Arendrup et al.(1993), Hinkula et al.(1994)]					
NOTES:						
	<ul style="list-style-type: none"> • P4/D10: Neutralizing and ADCC activity [Brolden et al.(1990)] • P4/D10: Variable domain sequenced and is identical to F58/H3 [Marks et al.(1992)] • P4/D10: Binding to native gp120 3 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore et al.(1993b)] • P4/D10: Primary isolates from different time points from one individual were not susceptible to neutralization by P4/D10 [Arendrup et al.(1993)] • P4/D10: Used for passive immunotherapy in four late-stage HIV-infected patients – the serum level of p24 did not decrease in any of these four – see also MAb F58/H3 [Hinkula et al.(1994)] 					
392 419-D	gp120(V3)	gp120(311-317)	IHIGPGR	L	HIV-1 infection	human(IgG _{1,λ})
References:	[Karwowska et al.(1992b), Gorny et al.(1993), Spear et al.(1993), Fontenot et al.(1995)]					
NOTES:						
	<ul style="list-style-type: none"> • 419-D: MN, NY5 and SF2 strain specific, does not cross-react with RF, CDC4, WM52 or HXB2 [Karwowska et al.(1992b)] • 419-D: Neutralizes MN – binds SF2: TYIGPGR [Gorny et al.(1993)] • 419-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG [Spear et al.(1993)] 					
393 537-D	gp120(V3)	gp120(313-317)	IGPGR	L	HIV-1 infection	human(IgG _{1,λ})
References:	[Karwowska et al.(1992b), Gorny et al.(1992), Gorny et al.(1993), VanCott et al.(1994), Fontenot et al.(1995)]					
NOTES:						
	<ul style="list-style-type: none"> • 537-D: Reacts with MN, NY5, CDC4, RF, WM52 and SF2, but does not cross-react with HXB2 [Karwowska et al.(1992b)] • 537-D: MN type specific neutralization observed – binds SF2, also IGPGR [Gorny et al.(1992), Gorny et al.(1993)] • 537-D: Moderate homologous neutralization, relatively rapid dissociation constant [VanCott et al.(1994)] 					
394 NM-01	gp120(V3 MN)	gp120(314-317)	GPGGR	L	IIIB MN	murine(IgG)
References:	[Ohno et al.(1991), Yoshida et al.(1997)]					
NOTES:						
	<ul style="list-style-type: none"> • NM-01: Resistance mutation selected by propagation of molecular cloned isolate in the presence of NM-01 [Yoshida et al.(1997)] 					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
395 447-52D	gp120(V3 MN)	gp120(314-317)	GPXR	L	HIV-1 infection	human(IgG _{3λ})

Donor: Dr. Susan Zolla-Pazner, NYU Med Center NY, NY, or Cellular Products Inc, Buffalo, NY, USA

References: [Gorny et al.(1992), Buchbinder et al.(1992), Karwowska et al.(1992b), Gorny et al.(1993), Keller et al.(1993), Cavacini et al.(1993a), Spear et al.(1993), Conley et al.(1994a), Laal et al.(1994), VanCott et al.(1994), Gorny et al.(1994), Moore et al.(1994a), Sattentau(1995), Fontenot et al.(1995), Saarloos et al.(1995), Zolla-Pazner et al.(1995), Zolla-Pazner & Sharpe(1995), Moore et al.(1995a), Moore & Ho(1995), Forthal et al.(1995), Jagodzinski et al.(1996), Trkola et al.(1996a), Sattentau(1996), D'Souza et al.(1997), Binley et al.(1997), Fouts et al.(1997), Hioe et al.(1997)]

NOTES:

- 447-52D: Also called 447/52-DII, 447-52-D, 447d, and 447-D (per. comm. S. Zolla-Pazner)
- 447-52D: Requires GPXR at the tip of the V3 loop – neutralizes a broad array of B clade lab isolates [Gorny et al.(1992)]
- 447-52D: 60-fold increase in neutralization potency when combined 1:1 with human MAb 588-D [Buchbinder et al.(1992)]
- 447-52D: Reacts with MN, NY5, CDC4, SF2, RF, WM52, and HXB2 [Karwowska et al.(1992b)]
- 447-52D: Neutralizes MN and IIIB: GPGGR, and binds SF2: GPGRR [Gorny et al.(1993)]
- 447-52D: Any of the residues ADGLMNQRS in the X position tolerated in peptides that react well with the antibody [Keller et al.(1993)]
- 447-52D: Additive neutralization of MN and SF2 when combined with CD4 binding site MAb F105 – supra-additive neutralization of RF [Cavacini et al.(1993a)]
- 447-52D: Complement mediated virolysis of IIIB, but not in the presence of sCD4 [Spear et al.(1993)]
- 447-52D: Requires GPxR at the tip of the V3 loop, common in B clade – neutralized primary isolates [Conley et al.(1994a)]
- 447-52D: Neutralization synergy in combination with CD4 binding domain MAb's [Laal et al.(1994)]
- 447-52D: GPGQ in MAL resulted in enhanced dissociation – GPGQ in CM234 or K14T did not bind – binding affected by identity of amino acids flanking GPGR core [VanCott et al.(1994)]
- 447-52D: Mild oxidation of carbohydrate moieties does not alter binding [Gorny et al.(1994)]
- 447-52D: Competition studies with human sera from seroconverting individuals showed that anti-CD4 BS antibodies can arise very early in infection, comparable or prior to anti-V3 antibodies [Moore et al.(1994a)]
- 447-52D: Called 447d – Formalin inactivation of virus at 0.1% formalin for 10 hours at 4 degrees was optimal for inactivation of virus while maintaining epitope integrity [Sattentau et al.(1995)]

MAb ID	MAb ID (cont.)	Properties and References
395 447-52D		<ul style="list-style-type: none"> • 447-52D: Called 447 – The tip of the V3 loop was presented in a mucin backbone – higher valency correlates with stronger affinity constant [Fontenot et al.(1995)] • 447-52D: Ab-mediated activation of complement on HIV+ cells is higher than Ab independent activation [Saarloos et al.(1995)] • 447-52D: Serotyping study using flow-cytometry – bound only to GPGR V3 loop tips [Zolla-Pazner et al.(1995)] • 447-52D: Neutralization of primary and prototype laboratory HIV-1 isolates using a resting cell assay enhances sensitivity [Zolla-Pazner & Sharpe(1995)] • 447-52D: Binding affected by identity of amino acids flanking GPGR core – poor breadth of primary virus neutralization [Moore et al.(1995a)] • 447-52D: Review: the V3 loop motif GPGR is not common outside subtype B isolates, MAb 19b is more cross-reactive [Moore & Ho(1995)] • 447-52D: Neutralizing (- complement), no ADCC activity, and no viral enhancing activity [Forthal et al.(1995)] • 447-52D: Called 447-52-D – The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – CRDS inhibits Called 447-52-D binding [Jagodzinski et al.(1996)] • 447-52D: Neutralizes JR-FL – strongly inhibits gp120 interaction with CCR-5 in a MIF-1β-CCR-5 competition study [Trkola et al.(1996a)] • 447-52D: Review: called 447-52-D – only four epitopes have been described which can stimulate a useful neutralizing response to a broad spectrum of primary isolates, represented by the binding sites of MAbs: 447-52-D, 2G12, Fab b12, and 2F5 [Sattentau(1996)] • 447-52D: In a multilaboratory blinded study, failed to consistently neutralize any of nine B clade primary isolates – many of these isolates had the GPGR motif at the apex of the V3 loop [D'Souza et al.(1997)] • 447-52D: An antibody with “intermediate” avidity as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)] • 447-52D: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 447-52D bound monomer, oligomer, and neutralized JRFL [Fouts et al.(1997)] • 447-52D: Tested using a resting cell neutralization assay [Hoe et al.(1997)] • 447-52D: Viral binding inhibition by 447-D was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
396 59.1	gp120(308-313 MN)	gp120(314-319)	GPGR _A F	L	V3 MN peptide	murine(IgG ₁)

Donor: Mary White-Scharf, Repligen Corporation

References: [D'Souza et al.(1991), White-Scharf et al.(1993), Potts et al.(1993), Ghiara et al.(1993), Bou-Habib et al.(1994), D'Souza et al.(1994), Seligman et al.(1996)]

NOTES:

- 59.1: Called R/V3-59.1 – potent neutralizing MAb [D'Souza et al.(1991)]
- 59.1: Epitope defined by peptide reactivity and binding affinity with amino acid substitutions – GPGR_AF [White-Scharf et al.(1993)]
- 59.1: Synergistic neutralization of MN when combined with sCD4 or the CD4BS MAb F105 [Potts et al.(1993)]
- 59.1: Crystal structure of a 24 amino acid peptide from the V3 loop bound to 59.1 Fab fragment i– contact residues IGPGR_AF [Ghiara et al.(1993)]
- 59.1: Greater affinity for T-cell tropic strain T-CSF than the primary isolate JR-CSF, from which T-CSF was derived [Bou-Habib et al.(1994)]
- 59.1: Multi-lab study for antibody characterization and assay comparison – neutralizes MN and IIIB [D'Souza et al.(1994)]
- 59.1: Competition ELISAs with serial deletions produced longer estimate of epitope length than x-ray crystallography or Alanine substitution, RIHIGPGR_AFYT_T, suggesting significance of non-contact residues [Seligman et al.(1996)]

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
397 50.1	gp120(V3 MN)	gp120(310-314)	RHIIG	L	V3 MN peptide	murine(IgG ₁)
Donor: Mary White-Scharf, RepliGen Corporation, Cambridge, MA References: [D'Souza et al.(1991), White-Scharf et al.(1993), Potts et al.(1993), Ghiaara et al.(1993), Rini et al.(1993), Bou-Habib et al.(1994), VanCott et al.(1994), Robert-Guroff et al.(1994), Moore et al.(1994b), VanCott et al.(1995), Fontenot et al.(1995), Seligman et al.(1996)]						
NOTES:						
	<ul style="list-style-type: none"> • 50.1: Called R/V3-50.1 – potent neutralizing MAb [D'Souza et al.(1991)] • 50.1: Epitope defined by peptide reactivity and changes affinity with amino acid substitutions – epitope RIHIGP [White-Scharf et al.(1993)] • 50.1: No synergistic neutralization of MN when combined with CD4BS MAb F105 – isotype stated to be IgG_{2a} [Potts et al.(1993)] • 50.1: Crystal structure of a 24 amino acid peptide from the V3 loop bound to 59.1 and 50.1 Fab fragments – epitope KRIHIGP [Ghiaara et al.(1993)] • 50.1: Crystal structure of V3 loop bound to 50.1 – light chain binds just to the left of GPG, heavy chain binds further to the left [Rini et al.(1993)] • 50.1: No neutralization of primary isolate JR-CSF – greater affinity for and neutralization of T cell tropic strain T-CSF, derived from JR-CSF [Bou-Habib et al.(1994)] • 50.1: Potent MN neutralization, slow dissociation rate [VanCott et al.(1994)] • 50.1: Chimeric MN V3 loop in an HXB2 background allows increased FACS signal, Ab affinity, and viral neutralization [Robert-Guroff et al.(1994)] • 50.1: Shows modest cross-reactivity among B clade gp120s, little outside B clade [Moore et al.(1994b)] • 50.1: Used to monitor HIV-1 Env expression in infected H9 cells [VanCott et al.(1995)] • 50.1: Competition ELISAs with serial deletions produced comparable estimate of epitope length to crystal structure and Alanine substitution – KRIHIGP [Seligman et al.(1996)] • 50.1: NIH AIDS Research and Reference Reagent Program: 1289 					
398 58.2	gp120(V3 MN)	gp120(312-319)	HIGPGRAY	L	MN V3 peptide	murine(IgG ₁)
	References: [White-Scharf et al.(1993), Potts et al.(1993), Moore et al.(1994b), Seligman et al.(1996)]					
NOTES:						
	<ul style="list-style-type: none"> • 58.2: Epitope defined by peptide reactivity and changes in affinity with amino acid substitutions – 4/7 primarily isolates were neutralized [White-Scharf et al.(1993)] • 58.2: Did not synergistically neutralize MN in combination with MAb F105 – there was synergistic neutralization when combined with sCD4 [Potts et al.(1993)] • 58.2: Modest cross-reactivity among B clade gp120s, little outside B clade – core epitope as I-IHIG [Moore et al.(1994b)] • 58.2: Competition ELISAs with serial deletions produced longer estimates of epitope length, RIIHIGPGRAY, than Alanine substitution, suggesting significance of non-contact residues [Seligman et al.(1996)] 					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
399 Nea 9301	gp120(V3 IIIB)	gp120(312-327)	RIGRGPGRRAFV TIGK-I			murine(unk)
	Donor: Dupont, commercial References: [Wagner et al.(1996)]					
	NOTES:					
• Nea 9301: [Wagner et al.(1996)]						
400 694/98-D	gp120(V3 IIIB)	gp120(316-319)	GRAF	L	HIV-1 infection	human(IgG _{1λ})
	Donor: Drs. S. Zolla-Pazner and M. Gorny, NYU Med Center NY, NY					
	References: [Gorny et al.(1992), Gorny et al.(1993), Cavacini et al.(1993a), Spear et al.(1993), Gorny et al.(1994), Laal et al.(1994), VanCott et al.(1994), Cook et al.(1994), VanCott et al.(1995), Zolla-Pazner et al.(1995), Forthal et al.(1995), Li et al.(1997)]					
	NOTES:					
• 694/98-D: Also called 694/98						
• 694/98-D: Antibody first described [Skinner et al.(1988b)]						
• 694/98-D: Type-specific lab isolate neutralization was observed [Gorny et al.(1992)]						
• 694/98-D: Neutralizes MN and IIIB (GRAF) – binds SF2 (GRAF) – binding reactivity: MN, IIIB, SF2, NY5, RF, CDC4, WM52 [Gorny et al.(1993)]						
• 694/98-D: Called 694-D – complement mediated virolysis of IIIB, but not in the presence of sCD4 [Spear et al.(1993)]						
• 694/98-D: 50% neutralization of HIV-IIIB at a concentration of 0.15μg/ml [Gorny et al.(1994)]						
• 694/98-D: Potent neutralization of IIIB – no neutralization synergy in combination with CD4 binding domain MAbs [Laal et al.(1994)]						
• 694/98-D: GRVY did not alter peptide binding – GRV1 and GQAW enhanced dissociation – GQVF and GQAL did not bind [VanCott et al.(1994)]						
• 694/98-D: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – V3 MAbs can inhibit gp120 binding to GalCer <i>in vitro</i> – binding of GalCer to gp120 inhibited but did not completely block MAb binding [Cook et al.(1994)]						
• 694/98-D: Human HIV-1 infected sera and MAb 694/98 have high reactivity to MN and RF infected H9 cells, but Genetech rec gp120 IIIB vaccine recipients do not [VanCott et al.(1995)]						
• 694/98-D: Serotyping study using flow-cytometry – bound GRAX bearing virus in 10/11 cases – somewhat conformation dependent [Zolla-Pazner et al.(1995)]						
• 694/98-D: ADCC activity, and no viral enhancing activity [Forthal et al.(1995)]						
• 694/98-D: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – could only achieve 50% neutralization alone – all Ab combinations tested showed synergistic neutralization – 694/98-D has synergistic response with MAbs F105, 15e, b12, 2F5, 17b, 2G12, and 48d, and with HIVIG [Li et al.(1997)]						

Mab ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
401 9205	gp120(V3 IIIB) Donor: NEN, Boston MA, commercial References: [Durda et al.(1990), Trujillo et al.(1993), Allaway et al.(1993), VanCott et al.(1994), Fontenot et al.(1995)]	gp120(317-319) core RAF	L	IIIB V3 Peptide	murine(IgG ₁)	
402 N11-20	gp120(V3 317-325) Donor: J. C. Maizie, Hybridiolab, Institut Pasteur References: [Valenzuela et al. (1998)]	gp120(314-322) GPGRAFVTI	L (LAI)	unk	murine(IgG _{1κ})	
403 902	gp120(V3 IIIB) Donor: Bruce Chesebro, Rocky Mountain National Laboratory, Montana References: [Chesebro & Wehrly(1988), Laman et al.(1993), Broder et al.(1994), Earl et al.(1994), Sakaiwa et al.(1997)]	gp120(315-326) PGRAFVTIGKIG	L	vaccinia-gp160 IIIB	murine(IgG _{1κ})	
404 IIIB-V3-01	gp120(V3 IIIB) Donor: Jon Laman References: [Laman et al.(1993)]	gp120(322-330) IKGKIGNMRQ	N V3-loop peptide	IIIB carboxy-terminus	murine(IgG ₁)	
NOTES:						
<ul style="list-style-type: none"> • 9205: Called NEA-9205, epitope RIQRGPGRAFVTIGK – Reacts with three human brain proteins of 35, 55, 110 kDa molecular weight – similar to 9284 [Trujillo et al.(1993)] • 9205: Synergy with combinations of CD4-based molecules in inhibition of HIV-1 Env mediated cell fusion [Allaway et al.(1993)] • 9205: Neutralizes IIIB but not MN – significantly slower dissociation constant for IIIB than MN [VanCott et al.(1994)] 						
NOTES:						
<ul style="list-style-type: none"> • N11-20: Also called 110-HI • N11-20: Neutralization of LAI in CEM cells by anti-V3 MAbs 110.4 and N11-20 is through inhibition of virus binding to the cell [Valenzuela et al.(1998)] 						
NOTES:						
<ul style="list-style-type: none"> • 902: Strain specific neutralization of HIV [Chesebro & Wehrly(1988)] • 902: Epitope may be partially masked or altered in the oligomeric molecule [Broder et al.(1994)] • 902: Used as a control in a study of the influence of oligomeric structure structure of Env in determining the repertoire of the Ab response [Earl et al.(1994)] • 902: NIH AIDS Research and Reference Reagent Program: 522 • 902: V3-BH10 peptide with loop-structure inhibits IL-2 induced T-cell proliferation, thought to be due to altering intracellular signaling, and MAb 908 can block the peptide inhibition [Sakaiwa et al.(1997)] 						
NOTES:						
<ul style="list-style-type: none"> • IIIB-V3-01: Specific for carboxy-terminal flank of the IIIB V3 loop – epitope is hidden native gp120, exposed on denaturation [Laman et al.(1993)] • IIIB-V3-01: UK Medical Research Council AIDS reagent: ARP3046 • IIIB-V3-01: NIH AIDS Research and Reference Reagent Program: 1726 						

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
405 9305	gp120(V3)	gp120		L		murine(unk)
	Donor: Du Pont, Wilmington DE					
	References: [McDougal et al.(1996)]					
406 D/6D1	gp120(V4 351-382 LAI)	gp120(349-381)	ASKLREQFGNNKTIIFKQSSGGDPEIVTHS-FN	N	Baculovirus-expressed gp120 LAI	murine(IgG ₁)
	References: [Bristow et al.(1994)]					
	NOTES:					
	• D/6D1: V4 MAb generated in a study of the humoral immune response to rgp120 and rgp160 [Bristow et al.(1994)]					
407 4D7/4	gp120(V4 361-380 LAI)	gp120(364-384)	IFKQSSGGDPEIVTHS-SFNCGG	Env glycopro		murine(IgG)
	Donor: S. Rajbar, NIBSC, UK					
	References: [Moore et al.(1994c)]					
	NOTES:					
	• 4D7/4: C3 region – the relative affinity for denatured/native gp120 is >10 [Moore et al.(1994c)]					
	• 4D7/4: UK Medical Research Council AIDS reagent: ARP3051					
408 36.1(ARP 329)	gp120(V4 362-381 LAI)	gp120(365-385)	FKQSSGGDPEIVTHS-FNCGGE	Env glycopro		murine(IgG)
			Moore et al.(1994c)]			
	References: [Thiriar et al.(1989), Moore et al.(1994c)]					
	NOTES:					
	• 36.1(ARP 329): in 36.1: The relative affinity for denatured/native gp120 is >30 – mutations 380 G/F, 381 E/P impair binding [Moore et al.(1994c)]					
	• 36.1(ARP 329): in 36.1: UK Medical Research Council AIDS reagent: ARP329					
409 C12	gp120(V4 362-381 LAI)	gp120(365-385)	FKQSSGGDPEIVTHS-FNCGGE	mis-folded LAI rgp160		murine(IgG ₁)
	Donor: George Lewis					
	References: [Moore & Ho(1993), Moore et al.(1994c), Abacioglu et al.(1994), Moore et al.(1994d)]					
	NOTES:					
	• C12: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]					
	• C12: The relative affinity for denatured/native gp120 is >30 – mutations 380 G/F, 381 E/P, and 384 Y/E impair binding – also binds GEFFYCNSTQLFNS, gp120(380-393 LAI) [Moore et al.(1994c)]					
	• C12: C3 region – epitope boundaries mapped by peptide scanning, core FNCGG [Abacioglu et al.(1994)]					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
410 110.D	gp120(C3 380-393 LAI) Donor: F. Traincard, Pasteur Institute, France References: [Moore et al.(1994c), Valenzuela et al.(1998)]	gp120(384-397)	G E F F Y C N S T Q L F N S	N	Env glycopro	murine(IgG)
411 B32	gp120(380-393 LAI) References: [Moore et al.(1994c), Abacioglu et al.(1994)]	gp120(384-397)	G E F F Y C N S T Q L F N S	mis-folded LAI rgp160	murine(IgG ₁)	
412 B2C	gp120(C3 HIV2ROD) References: [Matsushita et al.(1995)]	gp120	H Y Q (core)	L	Peptide	murine(unk)
413 2H1B	gp120(C3 370-376 HIV2ROD) References: [Matsushita et al.(1995)]	gp120(361-367)	R N I S F K A	N	Peptide	murine(unk)

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
414 2F19C	gp120(C3 HIV2ROD)	gp120	APGK(core)	N	Peptide	murine(unk)
	References: [Matsushita et al.(1995)]					
NOTES:	<ul style="list-style-type: none"> • 2F19C: Binds in WB, but binds poorly to Env on the cell surface [Matsushita et al.(1995)] 					
415 B15	gp120(V4 395-400 BH10)	gp120(394-399)	WFNSTW	mis-folded	LAI rgp160	murine(IgG _{2b})
	Donor: George Lewis					
	References: [Moore & Ho(1993), Moore et al.(1993b), Abacioglu et al.(1994)]					
NOTES:	<ul style="list-style-type: none"> • B15: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] • B15: Binds native BH10 gp120 with 5 fold less affinity than denatured – does not bind native or denatured MN gp120 [Moore et al.(1993b)] • B15: V4 region – epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)] 					
416 B34	gp120(V4 395-400 BH10)	gp120(394-399)	WFNSTW	mis-folded	LAI rgp160	murine(IgG _{2b})
	References: [Abacioglu et al.(1994)]					
NOTES:	<ul style="list-style-type: none"> • B34: V4 region – epitope boundaries mapped by peptide scanning [Abacioglu et al.(1994)] 					
417 7F11	gp120(397-439 IIIB)	gp120(396-440)	?	purified	gp120	murine(unk)
	References: [Lasky et al.(1987), Nilsen et al.(1996)]					
NOTES:	<ul style="list-style-type: none"> • 7F11: There is another MAb with this name that binds to integrase [Nilsen et al.(1996)] 					
418 5C2E5	gp120(C4 406-415 IIIB)	gp120(423-432)	QFINMWQEVK	purified	gp120	murine(unk)
	Donor: T. Gregory and R. Ward, Genetech, San Francisco					
	References: [Lasky et al.(1987), Cordell et al.(1991)]					
NOTES:	<ul style="list-style-type: none"> • 5C2E5: Blocks the gp120-CD4 interaction [Lasky et al.(1987)] • 5C2E5: Cross-competition with MAbs 5C2E5, ICR38.8f and ICR38.1a [Cordell et al.(1991)] 					

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MAb ID	Location	WEAU	Sequence	Neutralizing	ImmunoGen	Species(Isotype)
419 G3-211	gp120(C4 423-437 IIIB)	gp120(424-438)	NNMWQKVVGKAMYAP	L	virus derived IIIB gp120	murine(IgG1)
	References: [Sun et al.(1989)]					
	NOTES:					
	• G3-211: G3-211, 42, 299, 508, 519, 536, 537: Cross-react with diverse strains by immunofluorescence – blocks HIV binding to CD4+ cells – different neutralization efficiencies [Sun et al.(1989)]					
420 G3-537	gp120(C4 423-437 IIIB)	gp120(424-438)	NNMWQKVVGKAMYAP	L	virus derived IIIB gp120	murine(IgG1)
	References: [Sun et al.(1989), Ho et al.(1991b), McKeating et al.(1992b)]					
	NOTES:					
	• G3-537: G3-537, 211, 299, 508, 519, 536, 42: Cross-react with diverse strains by immunofluorescence – blocks HIV binding to CD4+ cells – different neutralization efficiencies [Sun et al.(1989)]					
	• G3-537: Weakly neutralizing – binds to a linear binding domain of gp120, NMWQEVEVGKAMYAPPISG [McKeating et al.(1992b)]					
421 polyclonal	gp120(CD4BS)	gp120(426-437)	NMWQEVEVGKAMYA	L	oral immunization – peptide plus cholera toxin adjuvant	murine(IgA)
	References: [Bukawa et al.(1995)]					
	NOTES:					
	• polyclonal: Polyclonal secretory IgA antibody raised by mucosal immunization is able to neutralize IIIB, SF2, and MN – HIV-1 neutralization may be due to the V3, CD4 or HPG30 component of the multicomponent peptide immunogen [Bukawa et al.(1995)]					
422 MO86/C3	gp120(C4 429-443)	gp120(430-444)	EVGKAMYAPPISGQI	rIIIIB Env 286-467	human(IgM)	
	References: [Ohlin et al.(1992)]					
	NOTES:					
	• MO86/C3: MO86: Generated through <i>in vitro</i> “immunization” of uninfected-donor lymphocytes [Ohlin et al.(1992)]					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunoigen	Species (Isotype)
423 G3-42	gp120(C4 429-438 BRU)	gp120(430-439)	EVGKAMYAPP	L	virus derived IIIB gp120	murine(IgG1)

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Sun et al.(1989), Moore et al.(1993b), Thali et al.(1993), Sattentau & Moore(1995), Jagodzinski et al.(1996), Moore & Sodroski(1996), Poinard et al.(1996a), Trkola et al.(1996a), Binley et al.(1997)]

NOTES:

- G3-42: Neutralization of IIIB but not RF [Sun et al.(1989)]
- G3-42: C4 region – binds HXB2 20mer KQIINMWQKVGVKAMYAPPIS, and SF-2 and MN gp120s – G3-42, G3-299 have lower affinity than G3-508, G3-519, and G3-536 – bound native gp120, not denatured – poor peptide binding, epitope spans V3-C4 regions – 433A/L, 435Y/H and 430V/S substitutions impaired binding, V3 loop insertion abolished binding [Moore et al.(1993b)]
- G3-42: Inhibits binding of CD4 inducible MAb 48d [Thali et al.(1993)]
- G3-42: Binds with higher affinity to monomer than to oligomer, slow association rate [Sattentau & Moore(1995)]
- G3-42: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – CRDS potently inhibits G3-42 binding – G3-42 epitope described as KVVGKAMYAPP [Jagodzinski et al.(1996)]
- G3-42: Inhibits binding of many anti-V3, -CD4 binding site, and -C4 region MAbs – enhances binding of some anti-V2 region MAbs [Moore & Sodroski(1996)]
- G3-42: Epitope described as KQIINMWQKVGVKAMYAPPIS – binding resulted in slight gp120 dissociation from virus and exposure of the gp41 epitope for MAb 50-69 [Poinard et al.(1996a)]
- G3-42: Called G3 42 – Does not inhibit gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study – described as V3-C4 discontinuous epitope [Trkola et al.(1996a)]
- G3-42: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)]

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunoigen	Species (Isotype)
424 G3-299	gp120(C4 429-438 BRU)	gp120(430-439)	EVGKAMYAPP	L	virus derived IIIB gp120	murine(IgG1)

Donor: M. Fung and Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Sun et al.(1989), Moore et al.(1993b), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Binley et al.(1997), Ditzel et al.(1997)]

NOTES:

- G3-299: Best neutralization of IIIB in panel of 7 MAbs that bind overlapping epitope [Sun et al.(1989)]
 - G3-299: C4 region – binds HXB2 20mer KQIINMWQKVGVKAMYAPPIS, and SF-2 and MN gp120s – G3-42, G3-299 lower affinity than G3-508, G3-519, and G3-536 – bound native gp120, not denatured – poor peptide binding, epitope spans V3-C4 regions – 433A/L, 435Y/H and 430V/S substitutions impaired binding, V3 loop cleavage or insertion abolished binding [Moore et al.(1993b)]
 - G3-299: Binds with higher affinity to monomer than to oligomer, slow association rate, although faster than other C4 MAbs tested, with more potent neutralization of lab strain [Sattentau & Moore(1995)]
 - G3-299: Discontinuous V3-C4 epitope, binding enhanced by a few anti-C1, anti-CD4 binding site, and V2 MAbs – binding reciprocally inhibited by anti-V3 MAbs – G3-229 enhances the binding of some anti-V2 MAbs [Moore & Sodroski(1996)]
 - G3-299: Epitope described as KQIINMWQKVGVKAMYAPPIS – binding resulted in slight gp120 dissociation from virus and exposure of the gp41 epitope for MAb 50-69 [Poignard et al.(1996a)]
 - G3-299: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)]

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Mab ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species (Isotype)
425 G3-508	gp120(C4 429-438 BRU)	gp120(430-439)	EVGKAMYAPP	L	virus derived IIIB gp120	murine(IgG1)
Donor: M. Fung and Tanox Biosystems Inc and David Ho, ADARC, NY						
References: [Sun et al.(1989), Thali et al.(1993), Moore et al.(1993b), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Trkola et al.(1997)]						
NOTES:						
• G3-508: Neutralization of IIIB and RF [Sun et al.(1989)]						
• G3-508: Inhibits binding of CD4 inducible MAbs 48d [Thali et al.(1993)]						
• G3-508: C4 region – binds HXB2 20mer KQIINMWQKVKGKAMYAPPIS, and SF-2 and MN gp120s – bound denatured with 10 fold greater affinity than native – 433A/L, 435Y/H and 430V/S substitutions impaired binding [Moore et al.(1993b)]						
• G3-508: Binds with higher affinity to monomer than to oligomer, slow association rate [Sattentau & Moore(1995)]						
• G3-508: Inhibits binding of some V3, C4 and CD4 binding site MAbs, enhances binding of V2 region MAbs [Moore & Sodroski(1996)]						
• G3-508: Binding resulted in slight gp120 dissociation from virus and exposure of the gp41 epitope for MAbs 50-69 [Poignard et al.(1996a)]						
• G3-508: Also called G3 508 – inhibits gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola et al.(1996a)]						
• G3-508: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)]						
426 G3-519	gp120(C4 429-438 BRU)	gp120(430-439)	EVGKAMYAPP	L	virus derived IIIB gp120	murine(IgG1)
Donor: Tanox Biosystems Inc and David Ho, ADARC, NY						
References: [Sun et al.(1989), Moore & Ho(1993), Moore et al.(1993b), D'Souza et al.(1994), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Binley et al.(1997)]						
NOTES:						
• G3-519: Best neutralization of RF in panel of 7 MAbs that bind overlapping epitope [Sun et al.(1989)]						
• G3-519: Neutralizes IIIB, is reactive with SF-2 gp120, mild inhibition of HIV-1+ sera binding to IIIB gp120 [Moore & Ho(1993)]						
• G3-519: C4 region – binds HXB2 20mer KQIINMWQKVKGKAMYAPPIS, and SF-2 and MN gp120s – bound denatured with 5 fold greater affinity than native – 433A/L, 435Y/H, 438P/R and 430V/S substitutions impaired binding [Moore et al.(1993b)]						
• G3-519: Included in a multi-lab study for antibody characterization, and binding and neutralization assay comparison, also binds IIIB: IIINMWQKVKGKAMYAPP [D'Souza et al.(1994)]						
• G3-519: Binds with higher affinity to monomer than to oligomer, slow association rate [Sattentau & Moore(1995)]						
• G3-519: Non-reciprocal enhanced binding in the presence of the C5 MAb 1C1 and the C1 MAb 135/9 – reciprocal enhanced binding with some V2 MAbs. Inhibited binding of the the presence of some C4, V3 and CD4 binding site MAbs [Moore & Sodroski(1996)]						
• G3-519: Epitope described as KVGIKAMYAPP – binding resulted in slight gp120 dissociation from virus but no significant exposure of the gp41 epitope for MAb 50-69 [Poignard et al.(1996a)]						
• G3-519: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)]						

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species (Isotype)
427 G3-536	gp120(C4 429-438 BRU)	gp120(430-439) EVGKAMYAPP	L	virus derived PIB gp120	murine(IgG1)	
Donor: Tanox Biosystems Inc and David Ho, ADARC, NY						
References: [Sun et al.(1989), Ho et al.(1991b), Cordell et al.(1991), McKeating et al.(1992b), Moore & Ho(1993), Moore et al.(1993b), Gorny et al.(1994), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a)]						
NOTES:						
• G3-536: Weak neutralization of PIB and RF – cross-react with diverse strains by immunofluorescence – blocks HIV binding to CD4+ cells – epitope:IIIMWQKVKGKAMYAP [Sun et al.(1989)]						
• G3-536: Cross-competition with Mabs 5C2E5, ICR38.8f and ICR38.1a [Cordell et al.(1991)]						
• G3-536: Weakly neutralizing – binds to a linear determinant in the CD4 binding domain of gp120 [McKeating et al.(1992b)]						
• G3-536: Neutralizes PIB, is reactive with SF-2 gp120, mild inhibition of HIV-1+ sera binding to PIB gp120 [Moore & Ho(1993)]						
• G3-536: C4 region – binds HXB2 20mer KQIINMWQKVKGKAMYAPPIS, and SF-2 and MN gp120s – bound denatured with 15 fold greater affinity than native – 433A/L, 435Y/H, 438P/R, and 430V/S substitutions impaired binding [Moore et al.(1993b)]						
• G3-536: Enhances binding of anti-V2 MAb 697-D [Gorny et al.(1994)]						
• G3-536: Binds with higher affinity to monomer than to oligomer, slow association rate [Sattentau & Moore(1995)]						
• G3-536: Inhibits binding of some V3, C4 and CD4 binding site Mabs, enhances binding of V2 region Mabs [Moore & Sodroski(1996)]						
• G3-536: Epitope described as KVKGKAMYAPP [Poignard et al.(1996a)]						
428 ICR38.1a	gp120(C4 429-438 BRU)	gp120(430-439) EVGKAMYAPP	L	rBH10 gp120 rat(IgG _{2b})		
References: [Cordell et al.(1991), McKeating et al.(1992b), Moore et al.(1992a), McKeating et al.(1992), McKeating et al.(1993b), McKeating et al.(1993a), Moore et al.(1993b), Jeffs et al.(1996)]						
NOTES:						
• ICR38.1a: Also called 38.1a						
• ICR38.1a: Weakly neutralizing – binds linear determinant in the CD4 binding domain – cross-competition with Mabs G3-36, 5C2E5, and ICR38.8f [McKeating et al.(1992b), Cordell et al.(1991)]						
• ICR38.1a: Unable to exert a synergistic effect in combination with V3 directed Mabs, in contrast to Mab 39.13g, that binds to a conformational epitope involved in CD4 binding [McKeating et al.(1992a)]						
• ICR38.1a: Studied in the context of a neutralization escape mutant [McKeating et al.(1993a)]						
• ICR38.1a: Unreactive with solid-phase decapeptide, competed in solution phase assay – ICR 38.1a and ICR 38.8f were initially reported to be two independent Mabs, but are actually subclones of the same MAb [Moore et al.(1993b)]						
• ICR38.1a: Called 38.1a – 10 to 20 fold increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs et al.(1996)]						
• ICR38.1a: UK Medical Research Council AIDS reagent: ARP388/ARP389						

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species (Isotype)
429 ICR38.8f	gp120(C4 429-438 BRU)	gp120(430-439)	EVGKAMYAPP	L	rBH10 gp120	rat(IgG _{2b})
References:	[Cordell et al.(1991)]					
NOTES:	<ul style="list-style-type: none"> ICR38.8f: Weakly neutralizing – binds linear determinant in the CD4 binding domain – cross-competition with ICR38.1a, 5C2E5, and G3-536 [Cordell et al.(1991)] ICR38.8f: ICR 38.1a and ICR 38.8f were initially reported to be to independent MAbs, but are actually subclones of the same MAb [Moore et al.(1993b)] 					
430 G45-60	gp120(C4 429-438 BRU)	gp120(432-441)	GKAMYAPPIS	L	virus derived IIIB gp[20]	murine(IgG ₁)
References:	[Sun et al.(1989), Moore et al.(1993b), Gorny et al.(1994), Moore & Sodroski(1996), Jagodzinski et al.(1996)]					
NOTES:	<ul style="list-style-type: none"> G45-60: C4 region – binds HXB2 20mer KQIINMWQKVKGKAMYAPPI, decapeptide flanking peptides also bound – bound equivalently to native and denatured gp120 – 433A/L and 435Y/H (not 430V/S) substitutions impaired binding [Moore et al.(1993b)] G45-60: Enhances binding of anti-V2 MAb 697-D [Gorny et al.(1994)] G45-60: Non-reciprocal enhancement of G45-60 binding by some C1 and C5 antibodies – reciprocal enhancement of some V2 region MAbs – reciprocal inhibition with many MAbs that bind to the V3, C4 and CD4 binding site regions [Moore & Sodroski(1996)] G45-60: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus CRDS inhibits G45-60 binding [Jagodzinski et al.(1996)] 					
431 1662	gp120(C4 IIIB)	gp120(434-440)	AMYAPP	N	poliovirus-antigen chimera	(unk)
References:	[McKeating et al.(1992b)]					
NOTES:	<ul style="list-style-type: none"> 1662: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)] 					
432 1663	gp120(C4 IIIB)	gp120(434-440)	AMYAPP	N	poliovirus-antigen chimera	(unk)
References:	[McKeating et al.(1992b)]					
NOTES:	<ul style="list-style-type: none"> 1663: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)] 					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
433 1664	gp120(C4 IIIB)	gp120(434-440)	AMYAPPI	N	poliovirus-antigen chimera	(unk)
	References: [McKeating et al.(1992b)]					
	NOTES:					
	• 1664: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]					
434 1697	gp120(C4 IIIB)	gp120(434-440)	AMYAPPI	N	poliovirus-antigen chimera	(unk)
	References: [McKeating et al.(1992b)]					
	NOTES:					
	• 1697: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]					
435 1794	gp120(C4 IIIB)	gp120(434-443)	AMYAPPISGQ	N	poliovirus env chimera	(unk)
	References: [McKeating et al.(1992b)]					
	NOTES:					
	• 1794: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]					
436 1804	gp120(C4 IIIB)	gp120(434-443)	AMYAPPISGQ	N	poliovirus env chimera	(unk)
	References: [McKeating et al.(1992b)]					
	NOTES:					
	• 1804: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]					
437 1807	gp120(C4 IIIB)	gp120(434-443)	AMYAPPISGQ	N	poliovirus env chimera	(unk)
	References: [McKeating et al.(1992b)]					
	NOTES:					
	• 1807: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
438 1808	gp120(C4 IIIB)	gp120(434-443)	AMYAPPISGQ	N	poliovirus env chimera	(unk)
	References: [McKeating et al.(1992b)]					
	NOTES:					
	• 1808: Did not bind to native gp120, epitope not exposed [McKeating et al.(1992b)]					
439 1795	gp120(CD4BS 425-441 III B)	gp120(326-342)	NMWQEVVGKAMYAPPIL	L	poliovirus env chimera	(unk)
		SG				
	References: [McKeating et al.(1992b)]					
	NOTES:					
	• 1795: CD4 binding site – weakly neutralizing – binding inhibited by WQEVGKAMYA, GKAM may be involved [McKeating et al.(1992b)]					
440 13H8	gp120(C4 412-453)	gp120(432-441)	GKAMYAPPIS	L	gp120 MN	murine(IgG)
	References: [Nakamura et al.(1992), Nakamura et al.(1993), Jeffs et al.(1996)]					
	NOTES:					
	• 13H8: Cross blocks 5C2 in IIIB-rgp160 ELISA – reactive with diverse strains in rgp120 ELISA [Nakamura et al.(1992)]					
	• 13H8: Bound diverse strains, neutralizing activity against MN [Nakamura et al.(1993)]					
	• 13H8: Binds V3 and C4 peptides (J. P. Moore, per. comm.)					
	• 13H8: 3 and 4.5 fold increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120, respectively [Jeffs et al.(1996)]					
441 M91	gp120(V5 C5 451-470 LAI)	gp120(463-472)	SNNESEITFRL	N	451 Env	rat(IgG _{2a})
	Donor: Fulvia di Marzo Veronese					
	References: [di Marzo Veronese et al.(1992), Moore et al.(1994c), Moore & Sodroski(1996), Ditzel et al.(1997)]					
	NOTES:					
	• M91: Immunoblot reactive, RIP negative, but precipitates deglycosylated gp120 – reacts with strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese et al.(1992)]					
	• M91: The relative affinity for denatured/native gp120 is 24 – mutation in position 470 P/L impairs binding [Moore et al.(1994c)]					
	• M91: 470 P/L impairs binding, but not 475 D/V, in contrast to CRA1 – some C2 mutations can enhance binding [Moore et al.(1994d)]					
	• M91: C5 region linear epitope, binds weakly to non-denatured monomeric gp120 – M91 binding was enhanced by 1C1, but 1C1 binding was inhibited by M91 – non-reciprocal binding enhancement of C1 and V2 antibodies – non-reciprocal binding inhibition of CD4 binding site antibodies [Moore & Sodroski(1996)]					

Mab ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
442 CRA1 (ARP 323)	gp120(V5-C5 451-470 LAI)	gp120(463-472)	NNESEIFRL	N	Env glycopro	murine(IgG)
Donor: M. Page, NIBSC, UK References: [Moore & Ho(1993), Moore et al.(1994d), Moore et al.(1994c), Moore & Sodroski(1996), Trkola et al.(1996a)]						
NOTES:						
<ul style="list-style-type: none"> • CRA1(ARP 323): CRA1: Also called CRA-1 • CRA1(ARP 323): CRA1: Bound preferentially to denatured IIIB and SF2 gp120 [Moore & Ho(1993)] • CRA1(ARP 323): CRA1: Some C5 mutations abrogate binding 470 P/L or G, 475 M/S, some C2 mutations enhance binding [Moore et al.(1994d)] • CRA1(ARP 323): CRA1: The relative affinity for denatured/native gp120 is 24 – C5 mutations 470 P/L or G, 475 M/S impairs binding to the native gp120 – only mutation 470 P/L impairs binding to denatured [Moore et al.(1994c)] • CRA1(ARP 323): CRA1: C5 region linear epitope, binds weakly to nondenatured monomeric gp120 – reciprocal binding inhibition with anti-C5 antibodies IC1 and M91 – non-reciprocal binding enhancement some C1 and V2 antibodies – non-reciprocal binding inhibition of some CD4 binding site antibodies [Moore & Sodroski(1996)] • CRA1(ARP 323): CRA1: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)] • CRA1(ARP 323): CRA1: UK Medical Research Council AIDS reagent: ARP323 						
443 9301	gp120(C5 471-490 LAI)	gp120(473-492)	GGGDMRDNWRS- ELYKKYKVVK	Env glycopro		murine(IgG)
Donor: Dupont, commercial References: [Skinner et al.(1988b), Moore & Ho(1993), Moore et al.(1994c), Moore et al.(1994d), Wagner et al.(1996)]						
NOTES:						
<ul style="list-style-type: none"> • 9301: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)] • 9301: The relative affinity for denatured/native gp120 is 19 [Moore et al.(1994d)] • 9301: Wagner et al. claim that Nea 9301 is anti-V3 – might they have meant Mab 9305? [Wagner et al.(1996)] 						
444 H11	gp120(C5 472-477 HXB2)	gp120(474-479?)	GGDMIRD?			murine(unk)
References: [Pincus et al.(1996)]						
NOTES:						
<ul style="list-style-type: none"> • H11: Binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing [Pincus et al.(1996)] 						

HIV Monoclonal Antibodies

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
445 M38	gp120(C5 490-508)	gp120(487-506)	KYKVVKEIPLGVAPT- KAKR	N	IIIB immunization	murine(unk)
	References: [Beretta et al.(1987), Grassi et al.(1991), Lopalco et al.(1993), DeSantis et al.(1994), Beretta & Dagleish(1994)]					
	NOTES:					
	• M38: Binds to gp120 and to a 80 kd human protein expressed on a small fraction of mononuclear cells in the lymph nodes [Beretta et al.(1987)]					
	• M38: Binds to the carboxy terminus of gp120, in a gp41 binding region, and also to denatured human HLAs (antigenic homology) [Lopalco et al.(1993)]					
	• M38: Infected individuals have HLA class I-gp120 cross-reactive antibodies [DeSantis et al.(1994)]					
446 1C1	gp120(C5 471-490 LAI)	gp120(473-492)	GGGDMRDNNWRSELYK- YKVVVK	Env glycopro	Env glycopro	murine (IgG)
	Donor: RepliGen Inc, Cambridge, MA, commercial					
	References: [Moore et al.(1994c), Moore et al.(1994d), VanCott et al.(1995), Moore & Sodroski(1996)]					
	NOTES:					
	• 1C1: The relative affinity for denatured/native gp120 is 15 [Moore et al.(1994c)]					
	• 1C1: C2 and V3 regions substitutions can influence binding [Moore et al.(1994d)]					
	• 1C1: Linear epitope not exposed on conformationally intact gp120 [VanCott et al.(1995)]					
	• 1C1: C5 region linear epitope, binds weakly to nondenatured monomeric gp120 – M91 binding was enhanced by 1C1, but 1C1 binding was inhibited by M91 – non-reciprocal binding enhancement of some C1 and V2 antibodies – non-reciprocal binding inhibition of some CD4 binding site antibodies [Moore & Sodroski(1996)]					
447 B221	gp120(C5 471-490 LAI)	gp120(473-492)	GGGDMRDNNWRSELYK- YKVVVK	Baculovirus-expressed mis-folded rgp160 IIIB:NL43, MicroGenSys	Baculovirus-expressed mis-folded rgp160 IIIB:NL43, MicroGenSys	murine(IgG ₁)
	Donor: Rod Daniels					
	References: [Moore & Ho(1993), Bristow et al.(1994), Moore et al.(1994c)]					
	NOTES:					
	• B221: Called 221 – bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]					
	• B221: MAbs generated in the context of a study of the humoral immune response to rgp120 and rgp160 – boundaries described as 443-462 of LAI [Bristow et al.(1994)]					
	• B221: The relative affinity for denatured/native gp120 is 12 – mutation 477 D/V impairs binding [Moore et al.(1994c)]					
	• B221: Called 221 – C2 and V3 substitutions influence binding [Moore et al.(1994d)]					
	• B221: UK Medical Research Council AIDS reagent: AR.P301					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
448 660-178	gp120(C5 471-490 LAI) Donor: G. Robey, Abbott Labs References: [Moore et al.(1994c), Moore et al.(1994d)] NOTES:	gp120(473-492)	GGGDMRDNWRSELYK- YKVVK	Env glycopro	murine(IgG)	
449 8C6/1	gp120(V5-C5 471-490 LAI) Donor: S. Ranjbar, NIBSC, UK References: [Moore et al.(1994c)] NOTES:	gp120(473-492)	GGGDMRDNWRSELYK- YKVVK	Env glycopro	murine(IgG)	
450 5F4/1	gp120(C5 471-490 LAI) Donor: S. Ranjbar, NIBSC, UK References: [Moore et al.(1994c)] NOTES:	gp120(473-492)	GGGDMRDNWRSELYK- YKVVK	Peptide	murine(unlk)	
451 3F5	gp120(C5 471-490 LAI) Donor: S. Nigida, NCI, USA References: [Moore et al.(1994c)] NOTES:	gp120(473-492)	GGGDMRDNWRSELYK- YKVVK	Env	murine(IgG)	
452 MO101/V3,C4	gp120(V3 314-323 + C5 494-503) References: [Ohlin et al.(1992)] NOTES:	gp120	GRAFVTIGKI + LGVAPTKAKR	pB1 (IIIB Env 286-467)	human(IgM)	

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
453 9201	gp120(C5 475-486 LAI) Donor: Du Pont References: [McDougal et al.(1996)]	gp120(473-484)	GGGDMRDNWRSF?	N		murine(unk)
	NOTES: • 9201: Does not neutralize LAI [McDougal et al.(1996)]					
454 W2	gp120(C5 472-491 LAI) Donor: D. Weiner, U. Penn., USA References: [Moore et al.(1994c)]	gp120(474-493) KVVKI	GGDMIRDNWRSELYKY-	Env		murine(IgG)
	NOTES: • W2: The relative affinity for denatured/native gp120 is 30 – mutation 485 K/V impairs binding [Moore et al.(1994c)]					
455 Chim 1	gp120(C5 492-498 HXB2) Donor: Pincus et al.(1996)	gp120(489-495)	KVVKEIF? References: [Pincus et al.(1996)]		humanized chimpanzee	
	NOTES: • Chim 1: binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing [Pincus et al.(1996)]					
456 RV110026	gp120(C5 491-500 LAI) Donor: Commercial, Olympus Inc References: [Moore et al.(1994c), Moore et al.(1994d)]	gp120(493-502)	IEPLGVAPTK	Peptide	human(unk)	
	NOTES: • RV110026: Preferentially binds SDS-DTT denatured gp120 (15 fold using R1/87 as capture reagent) [Moore et al.(1994c)]					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
457 110.1	gp120(C5 491-500 LAI)	gp120(493-502)	IEPLGVAPTK	N	BRU infected cell	murine(IgG _{1κ}) lysates
	Donor: Genetic Systems Corp, Seattle WA, E. Kinney-Thomas References: [Gosting et al.(1987), Linsley et al.(1988), Kinney Thomas et al.(1988), Pincus et al.(1991), Moore et al.(1994c), Cook et al.(1994), McDougal et al.(1996), Binley et al.(1997), Valenzuela et al.(1998)]					
	NOTES:					
	<ul style="list-style-type: none"> • 110.1: There is another antibody with this ID that binds to gp120, but at aa 200-217 [Pincus et al.(1996)] <ul style="list-style-type: none"> • 110.1: Referred to as 110-1 – does not inhibit CD4-gp120 binding or neutralize HIV-1 strains [Linsley et al.(1988)] • 110.1: Difference in the epitope: mapped to aa 421-429 (KQIIINMWQE), the T1 sequence – poor efficacy as an immunotoxin when linked to RAC [Pincus et al.(1991)] • 110.1: The relative affinity for denatured/native gp120 is 0.7 [Moore et al.(1994c)] • 110.1: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the carboxy-terminus of gp120 inhibit gp120 binding to GalCer but not as potently as anti-V3 MAbs – binding of GalCer to gp120 does not inhibit MAbs binding [Cook et al.(1994)] • 110.1: Does not neutralize HIV-1 LAI [McDougal et al.(1996)] • 110.1: A high avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)] • 110.1: Does effect LAI viral binding or entry into CEM cells [Valenzuela et al.(1998)] 					
458 GV1G2	gp120(494-499 IIIB)	gp120(496-501)	LGVAPT	gp120 complexed with mAb M77	gp120 complexed with mAb M77	murine(unk)
	References: [Denisova et al.(1996)]					
	NOTES:					
	<ul style="list-style-type: none"> • GV1G2: When anti-V3 MAbs M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes – MAbs GV12F6 and GV3H1 are homologous to GV1G2 and were generated in the same experiment [Denisova et al.(1996)] 					
459 722-D	gp120(C term 503-509)	gp120(505-511)	RRVVQRE	N	HIV-1 infection	human(IgG _{1κ})
	References: [Laal et al.(1994), Forthal et al.(1995)]					
	NOTES:					
	<ul style="list-style-type: none"> • 722-D: Not neutralizing alone, could synergize anti-CD4 binding site antibody neutralization [Laal et al.(1994)] • 722-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal et al.(1995)] 					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunocongen	Species(Isootype)
460 670-D	gp120(C term 503-509)	gp120(500-506)	PTKAKRR ?	N	HIV-1 infection	human(IgG ₁ λ)
	References: [Zolla-Pazner et al.(1995), Forthal et al.(1995)]					
	NOTES:					
	<ul style="list-style-type: none"> • 670-D: Group specific cross-clade binding in serotyping study using flow-cytometry [Zolla-Pazner et al.(1995)] • 670-D: Not neutralizing, positive ADCC activity, and no viral enhancing activity, numbering provided suggests epitope is RRVVQRE [Forthal et al.(1995)] 					
461 450-D	gp120(C term 475-486 or 503-509 BH10)	gp120(500-506)	PTKAKRR or RRVVQRE, or MRDNWWRSELYKY	N	HIV-1 infection?	human(IgG ₁ λ)
	Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY					
	References: [Durda et al.(1988), Karwowska et al.(1992a), Karwowska et al.(1992b), Spear et al.(1993), Laal et al.(1994), Gorny et al.(1994), Cook et al.(1994), Forthal et al.(1995), Manca et al.(1995), Li et al.(1997)]					
	NOTES:					
	<ul style="list-style-type: none"> • 450-D: Also called 450-D-3 and 450D • 450-D: Bound to MN, SF-2 and IIIB, but was not neutralizing [Karwowska et al.(1992a)] • 450-D: Did not mediate deposition of complement component C3 on HIV infected cells [Spear et al.(1993)] • 450-D: Not neutralizing alone, could synergize anti-CD4 binding site antibody neutralization [Laal et al.(1994)] • 450-D: Epitope is defined as PTKAKRR [Gorny et al.(1994)] • 450-D: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon - MAbs against the carboxy-terminus of gp120 do not inhibit gp120 binding to GalCer - binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)] • 450-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal et al.(1995)] • 450-D: Virions complexed to gp120 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca et al.(1995)] • 450-D: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env - 50% neutralization could not be achieved at a maximal concentration of 6 µg/ml [Li et al.(1997)] 					
462 750-D	gp120(C term 503-509)	gp120(500-506)	PTKAKRR	N	HIV-1 infection	human(IgG ₃ λ)
	References: [Forthal et al.(1995)]					
	NOTES:					
	<ul style="list-style-type: none"> • 750-D: Not neutralizing, positive ADCC activity, and no viral enhancing activity [Forthal et al.(1995)] 					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
463 120-1	gp120(C term 503-532)	gp120	?	N	Peptide	murine(IgM _κ)
	References: [Chanh et al.(1986), Dagleish et al.(1988)]					
464 858-D	gp120(C term 510-516)	gp120(507-513)	VVQREKRR	N	HIV-1 infection	human(IgG)
	References: [Zolla-Pazner et al.(1995), Forthal et al.(1995)]					
NOTES:						
	• 858-D: Group specific cross-clade binding in serotyping study, using flow-cytometry [Zolla-Pazner et al.(1995)]					
	• 858-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal et al.(1995)]					
465 989-D	gp120(C term)	gp120(507-513)	VVQREKRR	HIV-1 infection	human(IgG)	
	References: [Zolla-Pazner et al.(1995)]					
NOTES:						
	• 989-D: In serotyping study using flow-cytometry, showed B clade specificity, but only reacted with 7/11 B clade virus [Zolla-Pazner et al.(1995)]					
466 D7324	gp120(C term)	gp120	?	Peptide from the C-term	sheep(unk)	
	Donor: Aalto BioReagents Ltd, Dublin, Ireland					
	References: [Moore(1990), Sattentau & Moore(1991), Moore et al.(1993a), Moore et al.(1993b), Wyatt et al.(1995), Trkola et al.(1996a), Ditzel et al.(1997), Ugolini et al.(1997)]					
NOTES:						
	• D7324: Binding unaltered by gp120 binding to sCD4, in contrast to 110.5, 9284, 50-69 and 98-6 [Sattentau & Moore(1991)]					
	• D7324: Binds to the last 15 amino acids in gp120 – used for antigen capture ELISA [Wyatt et al.(1995)]					
	• D7324: Epitope in C5 – Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola et al.(1996a)]					
	• D7324: Used to capture gp120 onto solid phase for epitope mapping [Moore et al.(1993a), Moore et al.(1993b), Ditzel et al.(1997)]					
467 23A	gp120(C term)	gp120	?	N	?	(unk)
	Donor: J. Robinson, Tulane University, LA					
	References: [Thali et al.(1992a), Thali et al.(1993), Wu et al.(1996), Trkola et al.(1996a), Fouts et al.(1997)]					
NOTES:						
	• 23A: Called 2.3A – Did not block ability of gp120-sCD4 complexes to inhibit MIP-1 α binding – binds to gp41-binding domain [Wu et al.(1996)]					
	• 23A: C5 binding MAb – does not inhibit gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola et al.(1996a)]					
	• 23A: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric env binding – 23A bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)]					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
468 8F101	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS		sCD4-(rHXB2 gp120)-complex	murine(IgG)
	References: [Devico et al.(1995)]					
	NOTES:					
	• 8F101: MAbs specifically reactive to crosslinked gp120 and CD4 were derived (8F101, 8F102) – conformation dependent – competition studies indicate the epitope is immunogenic in infected humans [Devico et al.(1995)]					
469 8F102	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS		sCD4-(rHXB2 gp120)-complex	murine(IgG)
	References: [Devico et al.(1995)]					
	NOTES:					
	• 8F102: MAbs specifically reactive to crosslinked gp120 and CD4 were derived (8F101, 8F102) – conformation dependent – competition studies indicate the epitope is immunogenic in infected humans [Devico et al.(1995)]					
470 CG-10	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS	L	CD4/gp120 complex	murine(IgG ₁)
	Donor: Jonathan Gershoni, Tel Aviv University, Isreal					
	References: [Gershoni et al.(1993), Wu et al.(1996), Lee et al.(1997)]					
	NOTES:					
	• CG-10: CG-10 is also called CG10					
	• CG-10: Reacts exclusively with sCD4-gp120 complex, not with sCD4 or gp120 alone [Gershoni et al.(1993)]					
	• CG-10: Called CG10 – MIP-1 α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4, and MAAb CG10 does not block this inhibition [Wu et al.(1996)]					
	• CG-10: Called CG10 – Promotes envelope mediated fusion between both T-cell and macrophage tropic viruses and CD4+ lines; enhances viral infection and syncytium formation by lab strains – CG10 Fab fragment did not enhance fusion – MIP-1 α or β inhibition of infection of PM1 cells by macrophage tropic virus could be partially blocked by CG10 [Lee et al.(1997)]					
471 CG-4	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS	N	CD4/gp120 complex	murine(IgG ₁)
	Donor: Jonathan Gershoni, Tel Aviv University, Isreal					
	References: [Gershoni et al.(1993)]					
	NOTES:					
	• CG-4: Reacts with gp120 and sCD4-gp120 complex, not with sCD4 [Gershoni et al.(1993)]					
	• CG-4: Called CG4; does not enhance fusion, in contrast to CG10 [Lee et al.(1997)]					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
472 CG-9	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS	L	CD4/gp120 complex	murine(IgG ₁)
	References: [Gershoni et al.(1993)]					
NOTES:	• CG-9: Reacts preferentially with sCD4-gp120, also with sCD4, not with gp120 [Gershoni et al.(1993)]					
473 CG-25	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS	L	CD4/gp120 complex	murine(IgG ₁)
	References: [Gershoni et al.(1993)]					
NOTES:	• CG-25: Reacts preferentially with sCD4-gp120, also with sCD4, not with gp120 [Gershoni et al.(1993)]					
474 CG-76	gp120(dis gp120-CD4)	gp120(dis)	DISCONTINUOUS	L	CD4/gp120 complex	murine(IgG ₁)
	References: [Gershoni et al.(1993)]					
NOTES:	• CG-76: Reacts equally well with sCD4-gp120 and sCD4, but not with purified gp120 [Gershoni et al.(1993)]					
475 ID6	gp120(1-193 BH10)	gp120	UNDEFINED AMINO TERMINUS	?		murine(IgG ₁)
	References: [Ugen et al.(1993), Cook et al.(1994)]					
NOTES:	• ID6: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)]					
476 AD3	gp120(1-193 BH10)	gp120	UNDEFINED AMINO TERMINUS	?		murine(IgG ₁)
	References: [Ugen et al.(1993), Cook et al.(1994)]					
NOTES:	• AD3: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook et al.(1994)]					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
477 522-149	gp120(C1 dis)	DISCONTINUOUS	N	Env glycopro	(unk)	
Donor:	G. Robey, Abbott Inc.					
References:	[Moore & Sodroski(1996), Trkola et al.(1996a)]					
NOTES:	<ul style="list-style-type: none"> • 522-149: binding is enhanced by C5 antibodies M91 and 1C1 – mutual binding-inhibition with anti-C1 antibody 133/290 – binding is destroyed by a W/L (position 61, LAI) gp120 amino acid substitution – other C1 antibodies enhance binding to gp120 [Moore & Sodroski(1996)] • 522-149: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)] 					
478 MAG 45	gp120(C1 dis)	gp120(dis)	DISCONTINUOUS	N	sCD4-(rHXB2 gp120)-complex	murine(unk)
Donor:	C. Y. Kang, IDEC Inc					
References:	[Kang et al.(1994), Moore & Sodroski(1996)]					
NOTES:	<ul style="list-style-type: none"> • MAG 45: Only observed amino acid substitution that reduces binding: 88 N/P – does not bind to C1 region 20 mer peptides, tentative classification conformationally sensitive anti-C1 MAb [Kang et al.(1994)] • MAG 45: Reciprocal binding inhibition with anti-C1-C5 and -C1-C4 discontinuous MAbs – binding enhanced by anti-V3 5G11 – inhibits binding of anti-CD4 binding site MAbs [Moore & Sodroski(1996)] 					
479 MAG 95	gp120(C1 dis)	gp120(dis)	DISCONTINUOUS	N	sCD4-(rHXB2 gp120)-complex	murine(unk)
Donor:	C. Y. Kang, IDEC Inc					
References:	[Kang et al.(1994)]					
NOTES:	<ul style="list-style-type: none"> • MAG 95: Only observed amino acid substitution that reduces binding: 88 N/P – does not bind to C1 region 20 mer peptides, tentative classification conformationally sensitive anti-C1 MAb [Kang et al.(1994)] 					
480 MAG 97	gp120(C1 dis)	gp120(dis)	DISCONTINUOUS	N	sCD4-(rHXB2 gp120)-complex	murine(unk)
Donor:	C. Y. Kang, IDEC Inc					
References:	[Kang et al.(1994)]					
NOTES:	<ul style="list-style-type: none"> • MAG 97: Only observed amino acid substitution that reduces binding: 88 N/P – does not bind to C1 region 20 mer peptides, tentative classification conformationally sensitive anti-C1 MAb [Kang et al.(1994)] 					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
481 MAG 104	gp120(C1 dis)	gp120(dis)	DISCONTINUOUS	N	sCD4-(rHXB2 gp120)-complex	murine(unk)
Donor: C. Y. Kang, IDEC Inc References: [Kang et al.(1994)]						
NOTES:	• MAG 104: Only observed amino acid substitution that reduces binding: 88 N/P and 106 E/A – does not bind to C1 region 20 mer peptides, tentative classification conformationally sensitive anti-C1 MAb [Kang et al.(1994)]					
482 M90	gp120(C1 dis)	gp120(dis)	DISCONTINUOUS	N	451 Env	(IgG ₁)
Donor: Fulvia di Marzo Veronese References: [di Marzo Veronese et al.(1992), Devico et al.(1995), Moore & Sodroski(1996), Ditzel et al.(1997)]						
NOTES:	• M90: Reactive only with native gp120, so binds to a discontinuous epitope – reacts with multiple strains [di Marzo Veronese et al.(1992)] • M90: Reacted with both non-reduced (but not denatured) covalently cross-linked gp120-CD4 complex [Devico et al.(1995)] • M90: Reciprocal inhibition of binding of other anti-C1 MAbs – inhibits CD4 binding site MAbs – enhances binding of V2 MAbs G3-4 and SC258 [Moore & Sodroski(1996)]					
483 p7	gp120(C1 dis HXBc2)	gp120(dis)	DISCONTINUOUS	HIV infection	human Fab(IgG ₁)	
References: [Ditzel et al.(1997)]						
NOTES:	• p7: gp120 immobilized on solid phase by capture with sCD4 was used for selection of Fabs – three novel N-term Fabs were obtained that bind to similar epitopes, p7, p20, and p35 – a C1 W/S substitution at position 45 abolished binding, a Y/D at position 45 reduced binding, and C5 region substitutions 475 M/S and 493 P/K enhanced binding – compete with MAbs M85, M90 and 212A, but not M91 and G3-299 [Ditzel et al.(1997)]					
484 L19	gp120(C1 dis HXBc2)	gp120(dis)	DISCONTINUOUS	HIV infection	human Fab (IgG ₁)	
References: [Ditzel et al.(1997)]						
NOTES:	• L19: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for the selection of Fabs – six N-term Fabs, L19 L34, L35, L52, L59, and L69, were obtained that have a similar epitope to Fab p7 [Ditzel et al.(1997)]					

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Mab ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
485 L100	gp120(C1-C2 dis HXBc2)	gp120(dis)	DISCONTINUOUS		HIV infection	human Fab(IgG ₁)
References: [Ditzel et al.(1997)]						
NOTES:	<ul style="list-style-type: none"> • L100: gp120 immobilized on solid phase by capture with sCD4 and then masked with Fab p7 allowed selection of a new Fab, L100, with a novel specificity for C1 and C2 – gp120 C1 substitutions 69 W/L and 76 P/Y abolish L100 binding, and C2 substitutions 252 R/W, 256 S/Y, 262 N/T and 267 E/L abolish or strongly inhibit L100 binding – inhibits binding of MAbs M90 and G3-299, but not M85, 212A, and M91 [Ditzel et al.(1997)] 					
486 684-238	gp120(V2 dis)	gp120(dis)	DISCONTINUOUS	L	IIIIB gp120 from infected cells	murine(unk)
Donor: Gerry Robey, Abbott Laboratories						
References: [Moore et al.(1993a), Thali et al.(1993), Gorny et al.(1994), Ditzel et al.(1995), Moore & Sodroski(1996), Ditzel et al.(1997)]						
NOTES:	<ul style="list-style-type: none"> • 684-238: Also called 52-684-238 and 52-684 • 684-238: Specific for BH10 or HXB2, does not bind to MN, RF, or SF-2 gp120 – neutralizes BH10 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177FY/AT, 179/180LD/DL, 183/184PI/SG, and 192-194YSL/GSS [Moore et al.(1993a)] • 684-238: Weakly neutralizing, IC₅₀ • 684-238: Does not compete with IgG1b12, reciprocal inhibition with MAbs L39, L40, and L78 [Ditzel et al.(1995)] • 684-238: Limited reciprocal enhancement of binding with anti-V3 and C4 region antibodies – reciprocal inhibition with V2 region antibodies [Moore & Sodroski(1996)] 					
487 CRA-3	gp120(V2 dis)	gp120(dis)	DISCONTINUOUS	N	rBH10 gp120	murine(IgG _{2a})
Donor: Mark Page, NIBSC AIDS reagent project, Potters Bar, Herts, UK						
References: [Moore & Ho(1993), Moore et al.(1993a), Thali et al.(1993), Shotton et al.(1995), Moore & Sodroski(1996), Ditzel et al.(1997)]						
NOTES:	<ul style="list-style-type: none"> • CRA-3: Conformational, does not bind well to denatured gp120 [Moore & Ho(1993)] • CRA-3: specific for BH10 or HXB2, does not bind to MN, RF, or SF-2 gp120 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192-194 YSL/GSS – epitope probably involves stem of V1/V2 loop structure [Moore et al.(1993a)] • CRA-3: Many MAbs enhance binding, including some anti-C5, C1, V4, and C4 MAbs – enhances binding of only a small number of anti-V3 loop MAbs [Moore & Sodroski(1996)] • CRA-3: Called CRA3 – Same competition group as CRA6 [Shotton et al.(1995)] • CRA-3: UK Medical Research Council AIDS reagent: ARP324 					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
488 CRA-6	gp120(V1V2 dis)	gp120(dis)	DISCONTINUOUS	N	?	murine(unk)
	References: [Shotton et al.(1995)]					
	NOTES:					
	• CRA-6: Called CRA6 – same competition group as CRA-3 [Shotton et al.(1995)]					
489 CRA-4	gp120(V2 dis)	gp120(dis)	DISCONTINUOUS	L (HXB2)	rBH10 gp120	murine(IgG ₁)
	Donor: Mark Page, NIBS, MRC AIDS reagent repository, ARP 325					
	References: [McKeating et al.(1993b), Moore & Ho(1993), Moore et al.(1993a), Thali et al.(1993), Shotton et al.(1995), Moore & Sodroski(1996)]					
	NOTES:					
	• CRA-4: Also called CRA4					
	• CRA-4: Changes at residues 191/192/193 (YSL/GSS) within V2, 435 (Y/H) in C4, abrogate binding – type-specific neutralization [McKeating et al.(1993b)]					
	• CRA-4: Conformational, does not bind well to denatured gp120 [Moore & Ho(1993)]					
	• CRA-4: Specific for BH10 and HXB2, does not bind to MN, RF, or SF-2 gp120 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192-194 YSL/GSS [Moore et al.(1993a)]					
	• CRA-4: Cross-competes with MAbs 11/68b, 62c, 66c, 66a – similar to 66c and 66a – non-reciprocal inhibition by MAbs 12b, 60b and CRA-6 [Shotton et al.(1995)]					
	• CRA-4: The only MAbs that enhanced binding were anti-V3 MAbs 5G11 and anti-C1 MAb 135/9 binding – reciprocal inhibition of anti-V2 MAbs [Moore & Sodroski(1996)]					
	• CRA-4: UK Medical Research Council AIDS reagent: ARP325					
490 66a	gp120(V2 dis)	gp120(dis)	DISCONTINUOUS	L (HXB2)	rBH10 gp120	murine(IgG ₁)
	References: [Shotton et al.(1995)]					
	NOTES:					
	• 66a: Substitutions 176-177 FY/AT, 179-180 LD/DL, 183-184 PI/SG, and 191-193 YSL/GSS abrogate binding – same competition group as CRA4 [Shotton et al.(1995)]					
	• 66a: UK Medical Research Council AIDS reagent: ARP3074					
491 66c	gp120(V2 dis)	gp120(dis)	DISCONTINUOUS	L (HXB2)	rBH10 gp120	murine(IgG ₁)
	References: [Shotton et al.(1995)]					
	NOTES:					
	• 66c: Substitutions 176-177 FY/AT, 179-180 LD/DL, 183-184 PI/SG, and 191-193 YSL/GSS abrogate binding – same competition group as CRA4 [Shotton et al.(1995)]					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
492 11/68b	gp120(V1V2 dis)	gp120(dis)	DISCONTINUOUS	L (HXB2)	rBH10 gp120	rat(IgG1)
	Donor: Shotton and Dean					
	References: [McKeating et al.(1993b), Shotton et al.(1995)]					
	NOTES:					
	<ul style="list-style-type: none"> • 11/68b: Changes at residues 183/184 (PI/SG) within V2, 435 (Y/H) in C4, abrogate binding [McKeating et al.(1993b)] • 11/68b: 435 (Y/H) in C4 does not abrogate binding [John Moore, per comm, 1996] • 11/68b: Cross-competes with MAbs 62c, 66c, 66a, and CRA-4 – similar to MAb 62c – HXB2 neutralization escape mutant had a D/N substitution at residue 185 – non-reciprocal inhibition of binding of CRA-3 and CRA-6 [Shotton et al.(1995)] • 11/68b: UK Medical Research Council AIDS reagent: ARP3041 					
493 62c	gp120(V1V2 dis)	gp120(dis)	DISCONTINUOUS	N	rBH10 gp120	rat(IgG1)
	References: [Shotton et al.(1995)]					
	NOTES:					
	<ul style="list-style-type: none"> • 62c: Cross-competes with MAbs 11/68b, 66c, 66a, and CRA-4 – same cross-competition group as MAb 11/68b – non-reciprocal inhibition of binding of CRA-3 and CRA-6 – substitutions 176-177 FY/AT, 179-180 LD/DL, 183-184 PI/SG, and 191-193 YSL/GSS abrogate binding [Shotton et al.(1995)] • 62c: UK Medical Research Council AIDS reagent: ARP3075 					
494 SC258	gp120(V2 dis)	gp120(dis)	DISCONTINUOUS	L	IIIIB gp120 from infected cells	murine(unk)
	Donor: Gerry Robey, Abbott Laboratories					
	References: [Moore et al.(1993a), Thali et al.(1993), Gorry et al.(1994), Yoshiyama et al.(1994), Moore et al.(1994b), Moore et al.(1994c), Ditzel et al.(1995), Moore & Sodroski(1996), Trkola et al.(1996a), Ditzel et al.(1997)]					
	NOTES:					
	<ul style="list-style-type: none"> • SC258: Also called 52-581-SC258 – binds to BH10, MN, and RF gp120 – neutralizes BH10 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192-194 YSL/GSS [Moore et al.(1993a)] • SC258: HIV-1 RF V2 substitutions 177 Y/H and 179 L/P in the V2 loop of RF reduce affinity – 177 Y/H inhibits SC258 neutralization [Yoshiyama et al.(1994)] • SC258: Very poor reactivity with gp120 molecules outside of clade B [Moore et al.(1994b)] • SC258: Does not compete with IgG1b12 – reciprocal inhibition with MAbs L39, L40, and L78 [Ditzel et al.(1995)] • SC258: Several MAbs binding to various gp120 epitopes enhance binding, but the only MAb that SC258 enhanced binding of was anti-CD4 binding site MAb F91 – reciprocal inhibition with V2 region antibodies [Moore & Sodroski(1996)] • SC258: Does not inhibit gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study – listed as not neutralizing [Trkola et al.(1996a)] 					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species (Isotype)
495 110-B	gp120(V2 dis)	gp120(dis)	DISCONTINUOUS	n	BRU infected cell lysates	mouse(unk)
	Donor: Hybriddolabs, Institute Pasteur, Paris, France					
	References: [Moore et al.(1993a)]					
	NOTES:					
	• 110-B: specific for BH10, does not bind to MN, RF, or SF-2 gp120 – binding inhibited by deletion of the V2 loop, and the following amino acid substitutions: 168 K/I, 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192-194 YSL/GSS [Moore et al.(1993a)]					
496 L15	gp120(V2 dis)	gp120(dis)	DISCONTINUOUS	HIV infection	human(IgG ₁)	
	References: [Ditzel et al.(1997)]					
	NOTES:					
	• L15: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for selection of Fabs – 2 anti-V2 Fabs were obtained with very similar epitopes, L15 and L17 – deletions in V1 and V2 abolished binding, and rodent anti-V2 MAbs SC258, CRA3, G3-G4, G3-136, BAT-085, and 52-684 all compete with L15 [Ditzel et al.(1997)]					
497 L39	gp120(V2-CD4BS dis)	gp120(dis)	DISCONTINUOUS	N	HIV-1 infection	human(IgG _{1κ})
	References: [Ditzel et al.(1995)]					
	NOTES:					
	• L39: This Fab does not inhibit sCD4 binding, and but is inhibited by sCD4, probably due to conformational changes – it is competed by anti-V2 MAbs, and sensitive to amino acid substitutions in the V3 loop (similar patterns were observed for L39 and L78 gp120 amino acid substitutions enhancing or reducing binding) – does not compete with CD4BS MAbs, but is sensitive to amino acid changes at positions 368 and 370 – binding unaffected by deglycosylation – reciprocal inhibition with V2 MAbs SC258 and 684-238 – heavy and light chain variable region sequence is available [Ditzel et al.(1995)]					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species (Isotype)
498 L40	gp120(V2-CD4BS dis)	gp120(dis)	DISCONTINUOUS	N	HIV-1 infection	human(IgG _{1κ})
References: [Ditzel et al.(1995)]		NOTES:				
<ul style="list-style-type: none"> • L40: This Fab does not inhibit sCD4 binding, and but is inhibited by sCD4, probably due to conformational changes – it is competed by anti-V2 MAbs, and sensitive to amino acid substitutions in the V3 loop (similar patterns were observed for L40 and L78 gp120 amino acid substitutions enhancing or reducing binding) – does not compete with CD4BS MAbs, but is sensitive to amino acid changes at positions 368 and 370 – binding only partially affected by deglycosylation – reciprocal inhibition with V2 MAbs SC258 and 684-238 – heavy and light chain variable region sequence is available [Ditzel et al.(1995)] 						
499 L78	gp120(V2-CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG _{1κ})
References: [Ditzel et al.(1995)]		NOTES:				
<ul style="list-style-type: none"> • L78: Substitutions at V2: (152/153 GE/SM, 183/184 PI/SG, 191/193 YL/GS), 262 N/T, V3 (314 G/W), CD4BS (257 T/R, 368 D/R, 370 E/R) inhibit binding, and some C4 and C5 substitutions enhance binding – this Fab does not inhibit sCD4 binding, and but is inhibited by sCD4, probably due to conformational changes – it is competed by anti-V2 MAbs, and sensitive to amino acid substitutions in the V3 loop – does not compete with CD4BS MAbs, but is sensitive to amino acid changes at positions 368 and 370 – Fab neutralizes MN and LAI – binding unaffected by deglycosylation – reciprocal inhibition with V2 MAbs SC258 and 684-238 – heavy and light chain variable region sequence is available [Ditzel et al.(1995)] 						
500 L25	gp120(V2-CD4BS dis)	gp120(dis)	DISCONTINUOUS		HIV-1 infection	human(IgG ₁)
References: [Ditzel et al.(1997)]		NOTES:				
<ul style="list-style-type: none"> • L25: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for selection of Fabs – a single anti-V2 CD4 BS Fab was obtained with sensitivity to substitutions in the V2 and CD4 BS regions – rodent anti-V2 MAb SC258 competes with L25 [Ditzel et al.(1997)] 						

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
501 C11	gp120(C1-C5 dis)	gp120(dis)	DISCONTINUOUS	N	HIV-1 infection	human(unk)

Donor: J. Robinson, Tulane University, LA

References: [Robinson et al.(1992), Moore et al.(1994d), Moore & Sodroski(1996), Trkola et al.(1996a), Wu et al.(1996), Binley et al.(1997), Fouts et al.(1997)]

NOTES:

- C11: Also called c11
- C11: Mutations that inhibit binding: C1 (45 W/S, 88 N/P) – V5 (463 N/D) – and C5 (491 I/F, 493 P/K and 495 G/K) and enhance binding: C1 (36 V/L) – V1-V2 (152/153 GE/SM) – and Δ V1/V2/V3 [Moore et al.(1994d)]
- C11: Binding enhanced by anti-V3 MAbs 5G11 – reciprocal inhibition with anti-C1 MAbs [Moore & Sodroski(1996)]
- C11: Did not block ability of gp120-sCD4 complexes to inhibit MIP-1 α binding – binds to gp41-binding domain [Wu et al.(1996)]
- C11: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola et al.(1996a)]
- C11: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)]
- C11: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – C11 bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)]

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
502 212A	gp120(C1-C5 dis)	gp120(dis)	DISCONTINUOUS	N	HIV-1 infection	human(unk)
Donor:	J. Robinson, Tulane University, LA					
References:	[Robinson et al.(1992), Moore et al.(1994d), Moore & Sodroski(1996), Binley et al.(1997), Fouts et al.(1997), Ditzel et al.(1997)]					
NOTES:						
• 212A: Mutations that inhibit binding: C1 (45 W/S) and V5 (463 N/D) – and enhance binding: V2 (179/180 LD/DL) and C5 (495 G/K) [Moore et al.(1994d)]						
• 212A: Binding enhanced by anti-V3 MAb 5G11 – reciprocal inhibition with anti-C1 MAbs [Moore & Sodroski(1996)]						
• 212A: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)]						
• 212A: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 212A bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)]						
503 L81	gp120(C1-C5 dis)	gp120(dis)	DISCONTINUOUS	HIV infection		human(IgG ₁)
References:	[Ditzel et al.(1997)]					
NOTES:						
• L81: gp120 immobilized on solid phase by capture with anti-CD4 BS MAb L72 was used for selection of Fab – L81 binding is abolished by C1 substitution 45 W/S, C5 substitution 491 I/F, and C3 substitution L/A [Ditzel et al.(1997)]						

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
504 2G12	gp120(V3-C4 dis)	gp120(dis)	DISCONTINUOUS	L P	HIV-1 infection	human(IgG _{1,k})

Donor: Herman Katinger, Inst. Appl. Microbiol., Vienna, Austria

References: [Buchacher et al.(1994), Trkola et al.(1995), Moore & Ho(1995), McKeating et al.(1996), McKeating(1996), Trkola et al.(1996b), Moore & Sodroski(1996), Poignard et al.(1996b), Trkola et al.(1996a), Sattentau(1996), D'Souza et al.(1997), Mo et al.(1997), Binley et al.(1997), Fouts et al.(1997), Li et al.(1997), Moore & Trkola(1997), Mascola et al.(1997), Ugolini et al.(1997)]

NOTES:

- 2G12: Human MAb generated by electrofusion of PBLs from HIV-1+ volunteers with CB-F7 cells [Buchacher et al.(1994)]
- 2G12: Highly potent Cross-clade neutralizing activity [Trkola et al.(1995)]
- 2G12: Conformationally sensitive epitope destroyed by mutations altering the N-linked glycosylation sites near the base of the V3 loop and the amino-terminal flank of the V4 loop [Trkola et al.(1996b)]
- 2G12: Binding weakly enhanced by some anti-C1, -C4, -V3, and CD4 binding site MAbs – unusual in that 2G12 binding neither enhanced or inhibited the binding of other MAbs included in the study [Moore & Sodroski(1996)]
- 2G12: Review: binding site is distinct from CD4BS MAbs epitope and is unique among known gp120 MAbs, human or rodent [Moore & Ho(1995)]
- 2G12: Review: exceptional capacity to neutralize primary isolates in terms of both breadth and potency – one of three MAbs (IgG1b12, 2G12, and 2F5) generally accepted as having significant potency against primary isolates [Poignard et al.(1996b)]
- 2G12: Neutralizes JR-FL – inhibits gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola et al.(1996a)]
- 2G12: Neutralizes primary isolates, HXB2, and chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)]
- 2G12: Review: Only four epitopes have been described which can stimulate a useful neutralizing response to a broad spectrum of primary isolates, represented by the binding sites of MAbs: 447-52-D, 2G12, Fab b12, and 2F5 [Sattentau(1996)]
- 2G12: In a multilab evaluation of monoclonal antibodies, only IgG1b12, 2G12, and 2F5 could neutralize at least half of the 9 primary test isolates at a concentration of < 25 μ g per ml for 90% viral inhibition – neutralized 6 of 9 primary isolates [D'Souza et al.(1997)]

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MAb ID	MAb ID (cont.)	Information
504	2G12 (cont.)	<ul style="list-style-type: none">• 2G12: A JRCASF variant that was selected for IgG1b12 resistance remained sensitive to MAbs 2G12 and 2F5, for combination therapy [Mo et al.(1997)]• 2G12: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)]• 2G12: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 2G12 bound monomer, and weakly bound oligomer and neutralized JRFL [Fouts et al.(1997)]• 2G12: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 2G12 was a strong neutralizer of SHIV-vpu+ – all Ab combinations tested showed synergistic neutralization – 2G12 has synergistic response with MAbs 694/98-D (anti-V3), 2F5, F105, and b12 [Li et al.(1997)]• 2G12: Review: MAbs 2F5, 2G12 and IgG1b12 have potential for use in combination with CD4-IgG2 as an immunotherapeutic or immunoprophylactic – homologous MAbs to these are rare in humans and vaccine strategies should consider including constructs that may enhance exposure of these MAbs' epitopes [Moore & Trkola(1997)]• 2G12: Using concentrations of Abs achievable <i>in vivo</i>, the triple combination of 2F5, 2G12 and HIVIG was found to be synergistic to have the greatest breadth and magnitude of response against 15 clade B primary isolates [Mascola et al.(1997)]• 2G12: Viral binding inhibition by 2G12 was strongly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]• 2G12: UK Medical Research council AIDS reagent: ARP3030

Mab ID	Location	WEAU	Sequence	Neutralizing	ImmunoGen	Species(Iso-type)
505 SUMMARY CD4BS	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS			(unk)
		References: [Thali et al.(1993), Moore & Sodroski(1996)]				
NOTES:						
• SUMMARY CD4BS: Shared components of Mab epitopes and the discontinuous CD4 binding regions included Thr 257, Asp 368, Glu 370, Lys 421 through Trp 427 and Asp 457 [Thali et al.(1993)]						
• SUMMARY CD4BS: Anti-CD4 binding site antibodies (CD4BS) competitively inhibit CD4 binding to monomeric gp120, and they differ in precise dependence on gp120 residues, but generally require Asp-368 and Glu-370 [Moore & Sodroski(1996)]						
506 588-D	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG _{1κ})
	Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY					
	References: [Karwowska et al.(1992a), Buchbinder et al.(1992), Moore & Ho(1993), Jeffs et al.(1996)]					
NOTES:						
• 588-D: Also called 588						
• 588-D: Conformational – reactive with IIIB gp120 in RIP, but not WB assay [Karwowska et al.(1992a)]						
• 588-D: 4-fold increase in neutralization potency for 588-D when combined 1:1 with human MAb 447-D [Buchbinder et al.(1992)]						
• 588-D: Weak neutralization of IIIB – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)]						
• 588-D: Called 588 – slight, not significant increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs et al.(1996)]						
507 10/46c	gp120(CD4BS dis)	?	?	rgp120		rat(unk)
	References: [Cordell et al.(1991), Jeffs et al.(1996)]					
NOTES:						
• 10/46c: Increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs et al.(1996)]						
508 TH9	gp120(CD4BS)	?	?	L	?	human(IgG _{1κ}); human(IgG _{1κ})
	Donor: Michael Fung, Tanox Biosystem, USA					
	References: [D'Souza et al.(1995)]					
NOTES:						
• TH9: Found to neutralize MN, but not JRCSF, two B subtype primary isolates, or a D subtype primary isolate, by most labs in a multi-laboratory study involving 11 labs[D'Souza et al.(1995)]						

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
509 BM12	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(unk)
References:	[Kessler 2nd et al.(1995)]					
NOTES:	<ul style="list-style-type: none"> • BM12: Broad cross-clade neutralization of primary isolates – additive effect in combination with MAb 2F5 [Kessler 2nd et al.(1995)] 					
510 654-D	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	N	HIV-1 infection	human(IgG _κ)
Donor:	Susan Zolla-Pazner, NYU Med Center, NY, NY					
References:	[Karwowska et. al.(1993), Gorny et al.(1994), Stamatatos & Cheng-Mayer(1995), Li et al.(1997), Stamatatos et al.(1997)]					
NOTES:	<ul style="list-style-type: none"> • 654-D: Also called 654-30D and 654-30D • 654-D: Mild oxidation of carbohydrate moieties inhibits binding [Gorny et al.(1994)] • 654-D: Binds to HIV-1 SF128A and SF162 [Stamatatos & Cheng-Mayer(1995)] • 654-D: Called 654-30D – One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env [Li et al.(1997)] • 654-D: Anti-CD4 BS MAb 654-30D and IgG1b12 have comparable binding affinities, neither mediates gp120-virion dissociation, but IgG1b12 can neutralize SF128A and SF162 and 654-D cannot – 654-D actually enhances infection by both viruses in primary macrophages [Stamatatos et al.(1997)]; 					
511 S1-1	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG _{1,λ})
References:	[Lake et al.(1992), Moran et al.(1993), Wisniewski et al.(1996)]					
NOTES:	<ul style="list-style-type: none"> • S1-1: Neutralizes IIIB and MN without complement, and neutralizes RF and a clinical isolate with complement – binds to native but not denatured gp120 – inhibits sCD4-gp120 binding [Lake et al.(1992)] • S1-1: Heavy (V_HI) and light (V_λIII) chain sequenced – no enhancing activity – similar germline sequence to MAb 86, but very different activity [Moran et al.(1993)] • S1-1: S1-1 is V_H1 – V-region heavy chain usage was examined and a bias of enhanced V_H1 and V_H4, and reduced V_H3, was noted among HIV infected individuals [Wisniewski et al.(1996)] 					

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
512	559/64-D	gp120(CD4BS dis)	gp120(dis) DISCONTINUOUS	L	HIV-1 infection	human(IgG _{1κ})
	Donor:	Susan Zolla-Pazner, NYU Med Center, NY, NY				
	References:	[Karwowska et al.(1992a), McKeating et al.(1992), Spear et al.(1993), Forthal et al.(1995), Jeffs et al.(1996), Hioe et al.(1997)]				
	NOTES:					
	• 559/64-D: Also called 559					
	• 559/64-D: Conformational – reactive with IIIB gp120 in RIP, but not WB assay [Karwowska et al.(1992a)]					
	• 559/64-D: Did not mediate deposition of complement component C3 on HIV infected cells [Spear et al.(1993)]					
	• 559/64-D: Neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal et al.(1995)]					
	• 559/64-D: Called 559 – slight, not significant increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs et al.(1996)]					
	• 559/64-D: Used in the development of resting cell neutralization assay [Hioe et al.(1997)]					
513	428	gp120(CD4BS dis)	?		HIV-1 infection	human(unk)
	References:	[Karwowska et al.(1992a), Jeffs et al.(1996)]				
	NOTES:					
	• 428: Slight, not significant increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs et al.(1996)]					
514	558-D	gp120(CD4BS dis)	gp120(dis) DISCONTINUOUS	L	HIV-1 infection	human(unk)
	Donor:	Susan Zolla-Pazner, NYU Med Center, NY, NY				
	References:	[McKeating et al.(1992)]				
	NOTES:					
	• 558-D: Blocks gp120-CD4 binding – binds a panel of mutants all except for 256 S/Y and 262 N/T, which are probably conformationally disruptive [McKeating et al.(1992)]					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
515 448-D	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG _{1λ})
Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY						
References: [Karwowska et al.(1992a), McKeating et al.(1992), Spear et al.(1993), Laal et al.(1994), Forthal et al.(1995), Manca et al.(1995), Li et al.(1997)]						
NOTES:						
• 448-D: Also called 448D						
• 448-D: Conformational – reactive with IIIB gp120 in RIP, but not WB assay [Karwowska et al.(1992a)]						
• 448-D: Called 448D – blocks gp120-CD4 binding – substitutions at gp120 residues 88, 113, 117, 257, 368 and 370 reduce binding – epitope similar to rat MAbs 39.13 _g and 39.3b [McKeating et al.(1992)]						
• 448-D: Did not mediate deposition of complement component C3 on HIV infected cells [Spear et al.(1993)]						
• 448-D: Dissociation constant gp120 IIIB 0.029 – neutralizes IIIB, acts synergistically with anti-V3 Mab 447-52D [Laal et al.(1994)]						
• 448-D: Neutralizing activity, positive ADCC activity, and no viral enhancing activity [Forthal et al.(1995)]						
• 448-D: Virions complexed to gp120 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca et al.(1995)]						
• 448-D: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env [Li et al.(1997)]						
516 729-D	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG _{1κ})
Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY						
References: [Laal et al.(1994), D'Souza et al.(1997), Li et al.(1997)]						
NOTES:						
• 729-D: Also called 729-30D						
• 729-D: Dissociation constant gp120 IIIB 0.025 – neutralizes IIIB, acts synergistically with anti-V3 Mab 447-52D [Laal et al.(1994)]						
• 729-D: In a multilaboratory blinded study, failed to consistently neutralize any of nine B clade primary isolates – reported here to have a λ light chain, but originally reported in [Laal et al.(1994)] to be IgG _{1κ} [D'Souza et al.(1997)]						
• 729-D: Called 729-30D – one of 14 human MAbs tested for ability to neutralize chimeric SHIV-vpu+, which expressed HIV-1 IIIB env [Li et al.(1997)]						
517 654-D	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG _{1λ})
Donor: Susan Zolla-Pazner, NYU Med Center, NY, NY						
References: [Laal et al.(1994)]						
NOTES:						
• 654-D: Dissociation constant gp120 IIIB 0.008 – neutralizes IIIB, acts synergistically with anti-V3 Mab 447-52D [Laal et al.(1994)]						

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
518 HF1.7	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	purified anti-Leu-3a mAb	murine(IgM)
519 D20	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)
520 D60	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	vaccinia expressed oligomeric gp140 IIIB	murine(IgG)
521 50-61A	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG _κ)
522 48-16	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	N	HIV-1 infection	human(IgG _κ)
523 L41	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG _{1κ})

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
524 L28	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG _{I,κ})
References: [Ditzel et al.(1995)]						
NOTES:						
• L28: Substitutions at 257 T/R, 368 D/R, 370 E/Q, 475 M/S 102 E/L and 463 N/D reduce binding – binding was enhanced by removal of the V3 loop and by substitutions 45 W/S, 298 R/G, 381 E/P, 382 F/L, 420 I/R, 435 Y/H or Y/R – binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available [Ditzel et al.(1995)]						
525 L33	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG _{I,κ})
References: [Ditzel et al.(1995)]						
NOTES:						
• L33: binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available [Ditzel et al.(1995)]						
526 L42	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG _{I,κ})
References: [Ditzel et al.(1995)]						
NOTES:						
• L42: Substitutions at 257 T/R, 368 D/R, 370 E/R, 266 A/E and 477 D/V reduce binding – binding was significantly enhanced by 381 E/P and 382 F/L – binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available [Ditzel et al.(1995)]						
527 L52	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG _{I,κ})
References: [Ditzel et al.(1995)]						
NOTES:						
• L52: Binding is sensitive to deglycosylation – heavy and light chain variable region sequence is available [Ditzel et al.(1995)]						

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
528 GP13	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG1)
	References: [Schutten et al.(1993), Back et al.(1993), Bagley et al.(1994), Schutten et al.(1995a), Schutten et al.(1995b), Bolmstedt et al.(1996), Wisniewski et al.(1996), Schutten et al.(1996), Schutten et al.(1997)]					
	NOTES:					
	• GP13: Neutralized a broad range of HIV-1 strains from phylogenetically different subfamilies – the following gp120 amino acid substitutions strongly inhibit binding: 256(S/Y), 257(T/G), 262(N/T), 368(D/R or K), 370(E/R or Q or D), 384(Y/E) [Schutten et al.(1993)]					
	• GP13: Mutations in a neutralization resistant isolate obtained by passage of the IIIB isolate in chimpanzees reduced neutralization, but the escape was not as clear as seen with anti-V3 MAbs [Back et al.(1993)]					
	• GP13: Neutralizes IIIB – only slight inhibition of SI phenotype, and strong enhancement of NSI phenotype chimeric viruses, that incorporated different envs from the same donor [Schutten et al.(1995a)]					
	• GP13: Neutralizes T-cell adapted viruses but not the SI strain 16.2, despite high binding affinity [Schutten et al.(1995b)]					
	• GP13: Sera was obtained from guinea pigs vaccinated either with gp160, or with gp160 lacking N-linked glycans at N406, N448, and N463 – these sera could block equally well both the CD4 BS MAb GP13 and the V3 MAb F58/H3 [Bolmstedt et al.(1996)]					
	• GP13: GP13 is $V_H 5 - V$ -region heavy chain usage was examined and a bias of enhanced $V_H 1$ and $V_H 4$, and reduced $V_H 3$, was noted among HIV infected individuals [Wisniewski et al.(1996)]					
	• GP13: IIIB neutralizing MAbs <i>in vitro</i> fail to neutralize in a mouse model <i>in vivo</i> [Schutten et al.(1996)]					
	• GP13: Neutralized (50%) an SI-env chimeric virus and enhanced (>5 fold) an NSI-env chimeric virus [Schutten et al.(1997)]					
	• GP13: UK Medical Research council AIDS reagent: ARP3054					
529 GP44	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG1)
	References: [Schutten et al.(1993), Bagley et al.(1994), Wisniewski et al.(1996)]					
	NOTES:					
	• GP44: Exhibited a more restricted pattern of neutralizing activity than GP13 and GP68 – the following gp120 amino acid substitutions strongly inhibit binding: 256(S/Y), 257(T/G), 262(N/T), 368(D/R or K), 370(E/R or Q or D) [Schutten et al.(1993)]					
	• GP44: GP44 is $V_H 1 - V$ -region heavy chain usage was examined and a bias of enhanced $V_H 1$ and $V_H 4$, and reduced $V_H 3$, i was noted among HIV infected individuals [Wisniewski et al.(1996)]					
530 L72	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS			murine(unk)
	Donor: Dr. Hariharan, IDEC Pharmaceuticals Corp La Jolla, CA					
	References: [Ditzel et al.(1997)]					
	NOTES:					
	• L72: Used to bind gp120 to solid phase to select MAbs					

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Mab ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(IsoType)
531 GP68	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG1)
References: [Schutten et al.(1993), Klasse et al.(1993a), Bagley et al.(1994), Schutten et al.(1995a)]						
NOTES:						
<ul style="list-style-type: none"> • GP68: Neutralized a broad range of HIV-1 lab strains from phylogenetically different subfamilies – the following gp120 amino acid substitutions strongly inhibit binding: 117(K/W), 256(S/Y), 257(T/G), 262(N/T), 368(D/R or K), 370(E/R or Q), 384(Y/E), 435(Y/H) [Schutten et al.(1993)] • GP68: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – GP68 required markedly higher concentrations to neutralize the mutant than wild type [Klasse et al.(1993a)] • GP68: Neutralizes IIIB – only slight inhibition of SI phenotype, and strong enhancement of NSI phenotype chimeric viruses, that incorporated different envs from the same donor [Schutten et al.(1995a)] • GP68: GP68 is V_H1 – V-region heavy chain usage was examined and a bias of enhanced V_H1 and V_H4, and reduced V_H3, • GP68: UK Medical Research Council AIDS reagent: ARP3055 						
532 ICR 39.13g	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	rgp120 BH10	rat(IgG _{2b})
Donor: Jackie Cordell and C. Dean						
References: [Cordell et al.(1991), McKeating et al.(1992a), McKeating et al.(1992), McKeating et al.(1993b), Moore & Ho(1993), Thali et al.(1993), Klasse et al.(1993a), McLain & Dimmock(1994), Beretta & Dalgleish(1994), McKeating et al.(1996), Armstrong & Dimmock(1996), Klasse & Sattentau(1996)]						
NOTES:						
<ul style="list-style-type: none"> • ICR 39.13g: also known as ICR39.13g and 39.13g • ICR 39.13g: Cross-competes with MAbs ICR 39.3b and 15e [Cordell et al.(1991)] • ICR 39.13g: Binds to a conformational epitope involved in CD4 binding – exerts a synergistic effect in combination with V3 directed MAbs [McKeating et al.(1992a)] • ICR 39.13g: Neutralization activity against HXB10, RF, SF-2 and MN strains of HIV-1 [McKeating et al.(1993b)] • ICR 39.13g: Conformational, does not bind denatured gp120 – weak neutralization of IIIB – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)] • ICR 39.13g: Strongly inhibits CD4 inducible MAb 48d [Thali et al.(1993)] • ICR 39.13g: Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively – mediates neutralization with 2.3 molecules of IgG [McLain & Dimmock(1994)] • ICR 39.13g: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – ICR 39.13g required moderately higher concentrations to neutralize the mutant than wild type [Klasse et al.(1993a)] • ICR 39.13g: Called 39.13g Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)] • ICR 39.13g: Post-attachment neutralization mechanism, in contrast to MAbs 39.3b [Armstrong & Dimmock(1996)] • ICR 39.13g: Variants of LAI have differing neutralization susceptibility to 39.13g [Klasse & Sattentau(1996)] • ICR 39.13g: UK Medical Research Council AIDS reagent: ARP390 						

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
533 ICR 39.3b	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	rgp120 BH10	rat(IgG _{2b})

Donor: J. Cordell and C. Dean
References: [Cordell et al.(1991), McKeating et al.(1992), Moore et al.(1993b), McLain & Dimmock(1994), Armstrong & Dimmock(1996), Jeffs et al.(1996)]

NOTES:

- ICR 39.3b: also known as 39.3b and ICR39.3b
- ICR 39.3b: Cross-competes with MAbs ICR 39.13g and 15e [Cordell et al.(1991)]
- ICR 39.3b: Conformational, does not bind to denatured IIIB [Moore & Ho(1993)]
- ICR 39.3b: Kinetics of neutralization studied – no lag for 39.3b, while ICR 39.13g and ICR 41.1i have lags of 5 and 15 min respectively [McLain & Dimmock(1994)]
- ICR 39.3b: Neutralizes only if the antibody is added prior to the attachment of the virus to the cell, in contrast to 39.13g [Armstrong & Dimmock(1996)]
- ICR 39.3b: Called 39.3b – increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs et al.(1996)]
- ICR 39.3b: UK Medical Research Council AIDS reagent: ARP391

HIV Monoclonal Antibodies

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
534 15e	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG _{1κ})

Donor: J. Robinson, Tulane University, LA, and David Ho, ADARC, NY, NY

References: [Robinson et al.(1990), Thali et al.(1991), Cordell et al.(1991), Ho et al.(1991b), Koup et al.(1991), Ho et al.(1992), Wyatt et al.(1992), Thali et al.(1992a), Takeda et al.(1992), Moore & Ho(1993), Thali et al.(1993), Wyatt et al.(1993), Bagley et al.(1994), Thali et al.(1994), Cook et al.(1994), Moore et al.(1994b), Moore et al.(1994a), Sattentau & Moore(1995), Lee et al.(1995), McKeaning et al.(1996), Moore & Sodroski(1996), Poignard et al.(1996a), Trkola et al.(1996a), McDougal et al.(1996), Wisniewski et al.(1996), Binley et al.(1997), Fouts et al.(1997), Li et al.(1997)]

NOTES:

- 15e: Also called 1.5e and 15E – original paradigm for this type of antibody
- 15e: Broadly neutralizing, binds multiple strains, competes with CD4 for gp120 binding, DTT reduction of env abrogates binding – more potent blocking of gp120-sCD4 binding than MAbs G3-536 and G3-537 [Ho et al.(1991b)]
- 15e: Cross-competes with MAbs ICR 39.13g and ICR 39.3b [Cordell et al.(1991)]
- 15e: Binds to gp120 of HIV-1 IIIB, but not RF – mediates ADCC – deletion of the V3 loop from gp120 does not alter ADCC activity [Koup et al.(1991)]
- 15e: gp120 mutants that affect 15e epitope binding: 113, 257, 368, 370, 421, 427, 475 – four of these coincide with amino acids important for the CD4 binding domain [Ho et al.(1992)]
- 15e: Precipitation of Δ 297-329 env glycoprotein, with a deleted V3 loop, is much more efficient than precipitation of wild type [Wyatt et al.(1992)]
- 15e: Amino acid substitutions in HXB2 that strongly inhibit binding, similar to [Ho et al.(1992)], some additional, 88, 102, 117, 113, 257, 368, 370, 421, 427, 457, 470, 480 [Thali et al.(1992a)]
- 15e: Called N70-1.5e – does not enhance infection of HIV-1 IIIB and MN [Thali et al.(1992a)]
- 15e: Conformational, does not bind denatured gp120 – neutralizes IIIB – reactive with SF-2 gp120 – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)]
- 15e: Binding to Δ V1/2 and Δ V1/2/3 mutant glycoproteins is greater than binding to wildtype gp120 [Wyatt et al.(1993)]
- 15e: Called 15E – a neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – 15E neutralization was not affected by this mutation [Watkins et al.(1993)]
- 15e: Heavy chain is V_HIV, V2-1 – light chain is V_κI, Hum01/012. Compared to 21h and F105 [Bagley et al.(1994)]

MAb ID	534 15e (cont.)	<ul style="list-style-type: none"> • 15e: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs F105, 48d, 21h and 17b) [Thali et al.(1994)] • 15e: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – anti-CD4 MAbs moderately inhibit gp120 binding to GalCer, possibly through steric hindrance – binding of GalCer to gp120 inhibited but did not completely block 15e binding [Cook et al.(1994)] • 15e: Cross-reactive with gp120 proteins from clades B and D, less so with A and C, and not reactive with clade E and F [Moore et al.(1994b)] • 15e: Binds with higher affinity to monomer than to oligomer, moderate association rate [Sattentau & Moore(1995)] • 15e: The V4 and V5 domains are essential for 1.5e binding, in contrast to the V1, V2, and V3 loops [Lee et al.(1995)] • 15e: Called 1.5e – Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)] • 15e: gp120 binding enhanced by anti-V3 MAb 5G11 and anti-V2 MAb G3-136 – binding inhibited by other CD4 binding site MAbs, antibodies that bind to gp120 only when CD4 is bound, and CD4-IgG [Moore & Sodroski(1996)] • 15e: Anti-CD4BS MAbs 15e, 21h, and IgG1b12 did not cause gp120 dissociation from virus, or exposure of the gp41 epitope of MAb 50-69, in contrast to CD4i MAb 48d and anti-V3 neutralizing MAbs [Poignard et al.(1996a)] • 15e: Inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996)] • 15e: Neutralizes HIV-1 LAI less potently than V3 specific MAbs [McDougal et al.(1996)] • 15e: 15e is V_H4 – V-region heavy chain usage was examined and a bias of enhanced V_H1 and V_H4, and reduced V_H3, was noted among HIV infected individuals [Wisniewski et al.(1996)] • 15e: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)] • 15e: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 15e bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)] • 15e: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 15e could only achieve 50% neutralization, but could act synergistically with anti-V3 MAb 694/98-D to achieve 90% [Li et al.(1997)] • 15e: UK Medical Research Council AIDS reagent: ARP3016
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HIV Monoclonal Antibodies

MAb ID	Location	WEAU	Sequence	Neutralizing	ImmunoGen	Species(Isootype)
535 1125H	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L (MN)	HIV-1 infection	human(IgG _{1κ})
Donor:	Shermaine Tilley, Public Health Research Institute, USA					
References:	[Tilley et al.(1991b), Tilley et al.(1991a), Thali et al.(1992a), Wyatt et al.(1992), Pinter et al.(1993b), D'Souza et al.(1995), Warrier et al.(1996), Pincus et al.(1996)]					
NOTES:						
• 1125H: Binding to gp120 inhibited by CD4 – epitope is destroyed by reduction, but not by removal of N-linked sugars – potent neutralization of MN, RF, SF-2 and IIIB – neutralization synergy with anti-V3 MAb 4117C [Tilley et al.(1991a)]						
• 1125H: Amino acid substitutions in HXB2 that strongly inhibit binding: 88, 102, 117, 113, 257, 368, 370, 421, 427, 457, 470, 480 [Thali et al.(1992a)]						
• 1125H: Binding to soluble gp120 enhanced by the presence of an anti-V3 HuMAb, 41148D [Pinter et al.(1993b)]						
• 1125H: Precipitation of Δ 297-329 env glycoprotein, with has a deleted V3 loop, is much more efficient than precipitation of wild type [Wyatt et al.(1992)]						
• 1125H: Neutralization was MN specific – failed to neutralize JRCSF, and 2 B subtype and 1 D subtype primary isolates in a multi-laboratory study involving 11 labs [D'Souza et al.(1995)]						
• 1125H: Synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G [Warrier et al.(1996)]						
• 1125H: A panel immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus et al.(1996)]						
536 5145A	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(unk)
References:	[Pinter et al.(1993a), Warrier et al.(1996), Pincus et al.(1996)]					
NOTES:						
• 5145A: Potent and broadly cross-reactive neutralization of lab strains [Pinter et al.(1993a)]						
• 5145A: Synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G [Warrier et al.(1996)]						
• 5145A: A panel immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus et al.(1996)]						

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
537 21h	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG ₁)

Donor: J. Robinson, Tulane University, LA

References: [Ho et al.(1991b), Thali et al.(1992a), Ho et al.(1992), Wyatt et al.(1993), Moore & Ho(1993), Moore et al.(1994b), Moore et al.(1994a), Bagley et al.(1994), Thali et al.(1994), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Wisniewski et al.(1996), Binley et al.(1997), Fouts et al.(1997), Li et al.(1997), Ugolini et al.(1997)]

NOTES:

- 21h: Also called 2.1H
- 21h: Amino acid substitutions in HXB2 that inhibit binding, some shared with CD4 binding inhibition, 88, 113, 257, 368, 370, 421, 470, 480 [Thali et al.(1992a)]
- 21h: Binding to Δ V1/2 and Δ V1/2/3 mutant glycoproteins is greater than binding to wildtype gp120 [Wyatt et al.(1993)]
- 21h: Conformational, does not bind denatured gp120 – neutralizes IIIB – reactive with SF-2 gp120 – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)]
- 21h: Has strong cross-reactivity with gp120 monomers from most subtypes, A-F, with the least reactivity to clade E [Moore et al.(1994b)]
- 21h: Competition studies with human sera from seroconverting individuals showed that anti-CD4 BS antibodies can arise very early in infection, comparable or prior to anti-V3 antibodies [Moore et al.(1994a)]
- 21h: Heavy chain is V_H III, VDP-35 – light chain is V_{λ} IIa, Hum318. Compared to 15e and F105 [Bagley et al.(1994)]
- 21h: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs F105, 48d, 15e and 17b) [Thali et al.(1994)]
- 21h: Binds with higher affinity to monomer than to oligomer, moderate association rate [Sattentau & Moore(1995)]

HIV Monoclonal Antibodies

537 21h (cont.)

- 21h: Anti-CD4 binding site MAbs – reciprocal inhibition by anti-C1, -C4 and other anti-CD4 binding site antibodies – enhanced by some anti-V2 MAbs and anti-V3 MAb 5G11 – enhances binding of some anti-V3 and -V2 MAbs [Moore & Sodroski(1996)]
- 21h: Anti-CD4BS MAbs 15e, 21h, and IgG1b12 did not cause gp120 dissociation from virus, or exposure of the gp41 epitope of MAb 50-69, in contrast to CD4i MAb 48d and anti-V3 neutralizing MAbs [Poignard et al.(1996a)]
- 21h: 21h is V_H3 – V-region heavy chain usage was examined and a bias of enhanced V_H1 and V_H4, and reduced V_H3, was noted among HIV infected individuals [Wisniewski et al.(1996)]
- 21h: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)]
- 21h: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 21h bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)]
- 21h: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 50% neutralization could not be achieved at a maximal concentration of 67 µg/ml [Li et al.(1997)]
- 21h: Called 2.1H – Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating et al.(1996)]
- 21h: Viral binding inhibition by 21h strongly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)]
- 21h: UK Medical Research Council AIDS reagent: ARP3017

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
538 F105	gp120(CD4BS dis) Donor: Marshall Posner, Boston MA	gp120(dis)	DISCONTINUOUS	L	HIV-1 infection	human(IgG _{1κ})

References: [Posner et al.(1991), Thali et al.(1991), Thali et al.(1992a), Marasco et al.(1992), Wyatt et al.(1992), Posner et al.(1992b), Posner et al.(1992a), Moore & Ho(1993), Posner et al.(1993), Cavacini et al.(1993a), Cavacini et al.(1993b), Wyatt et al.(1993), Montefiori et al.(1993), Potts et al.(1993), Klasse et al.(1993a), Watkins et al.(1993), Bagley et al.(1994), Thali et al.(1994), Cook et al.(1994), Cavacini et al.(1994b), Cavacini et al.(1994a), Earl et al.(1994), Turbica et al.(1995), Posner et al.(1995), Cavacini et al.(1995), Sullivan et al.(1995), Khouri et al.(1995), Jagodzinski et al.(1996), Wolfe et al.(1996), McDougal et al.(1996), Wisniewski et al.(1996), Pincus et al.(1996), D'Souza et al.(1997), Li et al.(1997)]

NOTES:

- F105: First description of F105, binds topographically near the CD4-binding site – inhibits binding of free, infectious virions to uninfected HT-H9 cells, but does not react with virus adsorbed to uninfected HT-H9 cells – soluble rCD4 pre-bound to infected cells inhibits F105 binding – F105 inhibits infection of HT-H9 cells in standard neutralization assays with HIV-1 and MN strains [Posner et al.(1991)]
- F105: Neutralization escape mutants result from changes in amino acids in four discontinuous regions: C2, 256-262; C3, 386,370; C4, 421; and C5, 470, 475, 477, 482-484 of gp120 HXBc2 – anti-CD4 binding site (CD4BS) antibody [Thali et al.(1991)]
- F105: Amino acid substitutions that impair F105 neutralization inhibit gp120-CD4 interaction [Thali et al.(1992a)]
- F105: MAbs cDNA sequence – V_H4 V71-4 rearranged with a D_H D-D fusion product of dI4 and da4, and with J_{H5} – V_κ is from the *Human*325 germline gene joined with J_{κ2} [Marasco et al.(1992)]
- F105: Precipitation of Δ 297-329 env glycoprotein, with has a deleted V3 loop, is much more efficient than precipitation of wild type [Wyatt et al.(1992)]
- F105: F105 mediates ADCC against SF2 through the CD16+ population of PBMC – does not mediate complement-dependent cytotoxicity [Posner et al.(1992b)]
- F105: Significant enhancement of F105 binding to RF infected cells preincubated with V3-specific Mabs V3-2 and V3-1 [Posner et al.(1992a)]
- F105: Called F-105 – neutralizes IIIB – strong inhibition of HIV+ human sera binding to IIIB gp120 [Moore & Ho(1993)]

HIV Monoclonal Antibodies

MAb ID	538 F105 (cont.)	• F105: F105 to binds and neutralizes selected lab strains and 3/9 HIV-1 primary isolates – synergistic enhancement of neutralization by seropositive sera [Posner et al.(1993)]
		• F105: No neutralization of primary isolates observed (John Moore, pers comm)
		• F105: Additive MN or SF2 neutralization when combined with anti-V3 MAbs 447-52D and 257-D [Cavacini et al.(1993a)]
		• F105: Serum from all asymptomatic HIV-1 positive people tested block F105 binding, but only from 27% of symptomatic individuals [Cavacini et al.(1993b)]
		• F105: Binding to Δ V1/2 and Δ V1/2/3 mutant glycoproteins is 2.4- and 13-fold greater, respectively, than binding to wildtype gp120 [Wyatt et al.(1993)]
		• F105: Study of synergism between F105 and sera from vaccinated volunteers with V3-loop specific neutralization activity – 2/3 sera demonstrated neutralization synergy, and 3/3 binding/fusion-inhibition synergy [Montefiori et al.(1993)]
		• F105: Study of synergism of neutralization and binding comparing F105 and sCD4 with the V3 MAbs: 50.1, 59.1, 83.1, and 58.2 – synergy was observed, and the data suggest that binding of one ligand (F105) can increase the binding of the second (e. g. V3 loop MAbs) due to conformational changes [Potts et al.(1993)]
		• F105: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to a class of conformation sensitive neutralizing MAbs – required > 81 fold higher concentrations to neutralize the mutant than wild type [Klasse et al.(1993a)]
		• F105: A neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – F105 neutralization was not affected by this mutation [Watkins et al.(1993)]
		• F105: Comparison of MAb F105 sequences with those of MAbs 21h and 15e [Bagley et al.(1994)]
		• F105: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs 48d, 21h, 15e and 17b) [Thali et al.(1994)]
		• F105: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – anti-CD4 MAbs moderately inhibit gp120 binding to GalCer, possibly through steric hindrance – binding of GalCer to gp120 inhibited but did not completely block F105 binding[Cook et al.(1994)]
		• F105: Administered intravenously to four cynomolgus monkeys, plasma pharmacokinetics and biological activity tested [Cavacini et al.(1994b)]

MAb ID	
538 F105 (cont.)	<ul style="list-style-type: none"> • F105: Fab fragments show reduced capacity to neutralize IIIB, MN, and RF compared to intact IgG₁, suggesting bivalent interaction may be important in binding and neutralization [Cavacini et al.(1994a)] • F105: Used as a positive control for CD4 BS antibodies in a study of the influence of oligomeric structure of Env in determining the repertoire of the Ab response [Earl et al.(1994)] • F105: An immunoassay for titrating CD4BS serum antibody was developed using a gp120-coated solid phase and competition with MAb F105 – 109/110 French HIV-1+ sera and 51/56 HIV-1+ African sera had detectable CD4 BSAbs using this assay, demonstrating CD4 binding site conservation among diverse subtypes – CD4BS Abs were detected soon after seroconversion and persisted – 0/21 HIV-2+ sera reacted, indicating that the HIV-1 and HIV-2 CD4BS Abs are not cross-reactive [Turbica et al.(1995)] • F105: Eight patient phase Ia trial for use as an immunotherapeutic – no clinical or biochemical side effects observed, plasma levels \geq of 10 μg/ml maintained for 21 days [Posner et al.(1995)] • F105: Efficient neutralization of T-cell adapted lines HXBc2 and MN, no neutralization of primary isolates 89.6, ADA and YU2 – even some enhancement of infection of ADA and YU2 [Sullivan et al.(1995)] • F105: Biotinylated F105 was used for competition studies with Ab derived from pregnant HIV-1+ women – a correlation between maternal anti-CD4 BS Abs overlapping the F105 binding site and lack of HIV-1 transmission to infants was noted [Khouri et al.(1995)] • F105: Changing heavy chain from IgG₁ to IgG₃ increased neutralization efficiency [Cavacini et al.(1995)] • F105: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – deletion of the V3 loop results in less potent inhibition of F105 binding by CRDS – binding site of F105 described as 256-257 ST, 368-370 DPE, 421 K, and 470-484 PGGGDMRDNWWRSELY [Jagodzinski et al.(1996)] • F105: Phase I study – MAb clearance in plasma has a 13 day half-life [Wolfe et al.(1996)] • F105: Neutralizes HIV-1 LAI less potently than V3 specific MAbs [McDougal et al.(1996)] • F105: F105 is V_H4 – V-region heavy chain usage was examined and a bias of enhanced V_H 1 and V_H 4, and reduced V_H 3, was noted among HIV infected individuals [Wisniewski et al.(1996)] • F105: A panel immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus et al.(1996)] • F105: In a multilaboratory blinded study, failed to neutralize any of nine B clade primary isolates [D'Souza et al.(1997)] • F105: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – F105 could only achieve 50% neutralization alone – all Ab combinations tested showed synergistic neutralization – F105 has synergistic response with MAbs 694/98-D (anti-V3), 48d, 2F5, and 2G12, and also with HIVIG [Li et al.(1997)] • F105: NIH AIDS Research and Reference Reagent Program: 857

HIV Monoclonal Antibodies

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
539 IgG1b12	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L P	HIV-1 infection	human(IgG 1 κ)

Donor: D. Burton, Scripps Research Institute, La Jolla, CA, also J. Geltowsky, R. W. Johnson Pharmaceutical Research Inst. La Jolla, CA

References: [Burton et al.(1991), Barbas III et al.(1992), Roben et al.(1994), Burton et al.(1994), Moore et al.(1994b), Sattentau(1995), Moore et al.(1995a), Moore & Ho(1995), Parren et al.(1995), Trkola et al.(1995), Ditzel et al.(1995), Sullivan et al.(1995), Yang et al.(1997), Moore & Sodroski(1996), Gauduin et al.(1996), Poignard et al.(1996a), Poignard et al.(1996b), Trkola et al.(1996a), Sattentau(1996), McKeating(1996), D'Souza et al.(1997), Schutten et al.(1997), Mo et al.(1997), Fouts et al.(1997), Li et al.(1997), Kessler II et al.(1997), Moore & Trkola (1997), Stamatatos et al.(1997), Valenzuela et al.(1998), Ditzel et al.(1997), Ugolini et al.(1997)]

NOTES:

- IgG1b12: Fab b12 and IgG1b12 (also called IgG1-b12, IgG1 b12, IgGB12, and b4/12)
- IgG1b12: The original Fab fragment was derived from a combinatorial phage library from bone marrow of an HIV-1 positive individual [Burton et al.(1991)]
- IgG1b12: Anti-CD4 binding site Fab, potent neutralizing activity, greater affinity for a subpopulation of gp120 molecules suggested to be in a mature confirmation – mutations in gp120 that abrogate binding: 368 D/R or D/T, 370 E/R, and 477 D/V, of clone HXBc2 of LAI – sensitive to V1 and V2 substitutions [Roben et al.(1994)]
- IgG1b12: Very potent neutralization, of primary and lab strains, at concentrations that could be achieved by passive immunization – reduced binding with A,C, and D clade viruses relative to B clade, poor reactivity with E clade [Burton et al.(1994)]
- IgG1b12: Cross-reactive with some gp120s, (but not all), from clades A-D – not reactive with gp120 from clades E or F [Moore et al.(1994b)]
- IgG1b12: Formalin inactivation of virus at 0.1% formalin for 10 hours at 4 degrees was optimal for inactivation of virus while maintaining epitope integrity [Sattentau et al.(1995)]
- IgG1b12: Anti-CD4 binding site MAb – very potent neutralization of a number of primary isolates [Moore et al.(1995a)]
- IgG1b12: Complete protection against HIV-1 infection was achieved in hu-PBL-SCID mice by passive immunization with physiologically relevant doses [Parren et al.(1995)]
- IgG1b12: Called BM12 – broad cross-clade neutralization of primary isolates – additive neutralization in combination with MAb 2F5 [Kessler 2nd et al.(1995)]
- IgG1b12: Review: unusual properties for anti-CD4 BS MAb: sensitive to V2 substitutions, preferential recognition of the oligomer on the cell surface [Moore & Ho(1995)]

MAb ID	MAb ID (cont.)	Comments
539 IgG1b12	<ul style="list-style-type: none"> • IgG1b12: Could potently neutralize primary isolates from within clade B, but showed a slight reduction in efficacy outside of clade B [Trkola et al.(1995)] • IgG1b12: Because of this Fab's reduction in binding when the V2 loop is deleted and when aa 183/184 PI/SG substitutions are made [Roben et al.(1994)], competition studies were done with Fab L78 anti-V2 MAbs SC258 and 684-238; no competition was observed – b12 binding is glycosylation dependent and abrogated by denaturation. [Ditzel et al.(1995)] • IgG1b12: Fab b12 showed potent neutralization of T-cell-line-adapted strains, but much reduced neutralization of 3 primary isolates – 2 of the 3 primary isolates also had reduced binding affinity, but the third was as efficiently immunoprecipitated as HXBc2 [Sullivan et al.(1995)] • IgG1b12: Saturation mutagenesis of the complementarity-determining region and optimization strategies were used to create very high affinity versions of this Fab – increased affinity was dominated by a slowing of the off rate [Yang et al.(1997)] • IgG1b12: Potent neutralizing <i>ex vivo</i> of virus taken directly from plasma of HIV-1 infected individuals – little correlation between neutralization sensitivity of passaged virus and plasma derived virus – more effective than MAb 19b [Gauduin et al.(1996)] • IgG1b12: Review: Unique among anti-CD4BS MAbs in terms of being potent against both lab adapted virus and primary isolates – one of three MAbs (IgG1b12, 2G12, and 2F5) generally accepted as having significant potency against primary isolates [Poignard et al.(1996b)] • IgG1b12: Anti-CD4BS MAbs 15e, 21h, and IgG1b12 did not cause gp120 dissociation from virus, or exposure of the gp41 epitope of MAb 50-69, in contrast to CD4i MAb 48d and anti-V3 neutralizing MAbs [Poignard et al.(1996a)] • IgG1b12: Neutralizes JR-FL – inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)] • IgG1b12: Review: Only four epitopes have been described which can stimulate a useful neutralizing response to a broad spectrum of primary isolates, represented by the binding sites of MAbs: 447-52-D, 2G12, Fab b12, and 2F5 [Sattentau(1996)] • IgG1b12: In a multilab evaluation of monoclonal antibodies, only IgG1b12, 2G12, and 2F5 could neutralize at least half of the 9 primary test isolates at a concentration of < 25 μg per ml for 90% viral inhibition – IgG1b12 failed to neutralize only 1/9 primary isolates, although there was some variation between test sites [DSouza et al.(1997)] 	

HIV Monoclonal Antibodies

MAb ID	MAb ID (cont.)	Description
539	IgG1b12 (cont.)	<ul style="list-style-type: none"> • IgG1b12: Inhibited some SI- and NSI-env chimeric viruses but enhanced one NSI-env chimeric virus 3 fold [Schutten et al.(1997)] • IgG1b12: JRCSF was cultured in the presence of IgG1b12 until a 100-fold resistance to neutralization was selected – resistance was due to three changes: V2 substitution D182N and C3 substitution P365L conferred resistance, and V2 D164N was also required for a viable virus – IgG1b12 resistant virus remained sensitive to MAbs 2G12 and 2F5 [Mo et al.(1997)] • IgG1b12: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – IgG1b12 bound monomer, oligomer, and neutralized JRFL [Fouts et al.(1997)] • IgG1b12: b12 was used in its IgG1 form – of 14 human MAbs, the most potent neutralizer of SHIV-vpu+, which expressed HIV-1 IIIB env – all Ab combinations tested showed synergistic neutralization – b12 has a synergistic response with MAbs 694/98-D (anti-V3), 2F5, and 2G12 [Li et al.(1997)] • IgG1b12: 35 primary isolates were tested and all were neutralized by IgG1b12 (including 4, UG270, RW92/026, ZB20, and 301727 which been had reported as not neutralized by IgG1b12 [Trkola et al.(1995)]) – IgG1b12 could neutralize even when added after the virus to the culture – selection for 400-fold increased affinity did not enhance neutralization by antibody – IgG1b12 was more potent with greater breadth than MAb 2F5 [Kessler II et al.(1997)] • IgG1b12: Review: MAbs 2F5, 2G12 and IgG1b12 have potential for use in combination with CD4-IgG2 as an immunotherapeutic or immunoprophylactic – homologous MAbs to these are rare in humans and vaccine strategies should consider including constructs that may enhance exposure of these MAbs' epitopes [Moore & Trkola(1997)] • IgG1b12: MAb was slightly more efficient at neutralization than Fab – inhibits viral binding to cells and viral entry – doesn't effect CD4-independent binding to T-cells [Valenzuela et al.(1998)] • IgG1b12: Viral binding inhibition by IgG1b12 strongly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)] • IgG1b12: UK Medical Research Council AIDS reagent: ARP3065 • IgG1b12: NIH AIDS Research and Reference Reagent Program: 2640

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
540 F91	gp120(CD4BS dis) gp120 [Moore & Ho(1993)]	gp120(dis) DISCONTINUOUS				(unk)
	Donor: J. Robinson, Tulane University, LA References: [Moore & Ho(1993), Moore et al.(1994b), Moore & Sodroski(1996), Fouts et al.(1997)]					
	NOTES:					
	<ul style="list-style-type: none"> • F91: Called F-91 – neutralizes IIIB – reactive with SF-2 gp120 – strong inhibition of HIV+ human sera binding to IIIB • F91: Has strong cross-reactivity with gp120 monomers from most subtypes, A-F [Moore et al.(1994b)] • F91: Unusual pattern of reciprocal enhancement with several anti-V2 and V3 directed MAbs – reciprocal inhibition of other CD4BS MAbs [Moore & Sodroski(1996)] • F91: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – F91 bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)] 					
541 HT6	gp120(CD4BS dis) gp120(dis) [Moore et al.(1994b), Moore et al.(1995a), Fouts et al.(1997)]	gp120(dis) DISCONTINUOUS	L (weak)	HIV-1 infection	human(unk)	human(unk)
	Donor: Ciba-Geigy AG (Basel, Switzerland) References: [Moore et al.(1995a), Fouts et al.(1997)]					
	NOTES:					
	<ul style="list-style-type: none"> • HT6: HT6, HT5, and HT7 are also known as 205-46-9, 205-42-15, and 205-43-1 • HT6: Despite highly cross-reactive binding to many primary and T-cell adapted viral strains, only weakly neutralizes IIIB and MN [Moore et al.(1995a)] • HT6: 205-46-9 was cross-reactive across clades A-F, 205-43-1 was not [Moore et al.(1994b)] • HT6: MAbs IgG1b12, HT5, HT6, and HT7 cross-compete for binding to monomeric gp120, bind equally well, inhibit gp120-sCD4 interactions, but only IgG1b12 neutralizes JRFL [Fouts et al.(1997)] 					
542 HT5	gp120(CD4BS dis) gp120(dis) [Moore et al.(1994b), Moore et al.(1995a), Fouts et al.(1997)]	gp120(dis) DISCONTINUOUS	L (weak)	HIV-1 infection	human(unk)	human(unk)
	Donor: Ciba-Geigy AG (Basel, Switzerland) References: [Moore et al.(1995a), Fouts et al.(1997)]					
	NOTES:					
	<ul style="list-style-type: none"> • HT5: HT6, HT5, and HT7 are also known as 205-46-9, 205-42-15, and 205-43-1 (John Moore, per comm) • HT5: Despite highly cross-reactive binding to many primary and T-cell adapted viral strains, only weakly neutralizes IIIB and MN [Moore et al.(1995a)] • HT5: 205-46-9 was cross-reactive across clades A-F, 205-43-1 was not [Moore et al.(1994b)] • HT5: MAbs IgG1b12, HT5, HT6, and HT7 cross-compete for binding to monomeric gp120, bind equally well, inhibit gp120-sCD4 interactions, but only IgG1b12 neutralizes JRFL [Fouts et al.(1997)] 					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isootype)
543 HT7	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L (IIB)	HIV-1 infection	human(unk)
Donor:	Ciba-Geigy AG (Basel, Switzerland)					
References:	[Moore et al.(1994b), Moore et al.(1995a), Fouts et al.(1997)]					
NOTES:						
• HT7: HT6, HT5, and HT7 are also known as 205-46-9, 205-42-15, and 205-43-1						
• HT7: Despite highly cross-reactive binding to many primary and T-cell adapted viral strains, only neutralizes IIB well, with sporadic weak neutralization of other isolates [Moore et al.(1995a)]						
• HT7: 205-46-9 was cross-reactive across clades A-F, 205-43-1 was not [Moore et al.(1994b)]						
• HT7: MAbs IgG1b12, HT5, HT6, and HT7 cross-compete for binding to monomeric gp120, bind equally well, inhibit gp120-sCD4 interactions, but only IgG1b12 neutralizes JRFL [Fouts et al.(1997)]						
544 MAG 55	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	sCD4-(rHXB2 gp120)-complex	murine(unk)
Donor:	C. Y. Kang, IDEC Inc					
References:	[Kang et al.(1994), Moore & Sodroski(1996)]					
NOTES:						
• MAG 55: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 421 K/L, 470 P/L, 475 M/S, 477 D/V – neutralizes MN, IIB and RF [Kang et al.(1994)]						
• MAG 55: Called #55 – binding reciprocally inhibited by other anti-CD4 binding site MAbs, and by some C1-C5 MAbs – binding enhanced by anti-V3 MAbs G110.5 and anti-V2 MAbs G3-136 and G3-4 – enhances binding of many anti-V3 and -V2 MAbs. [Moore & Sodroski(1996)]						
545 MAG 72	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	sCD4-(rHXB2 gp120)-complex	murine(unk)
Donor:	C. Y. Kang or Dr. Hariharan, IDEC Pharmaceuticals Corp, La Jolla, CA					
References:	[Kang et al.(1994), Ditzel et al.(1997)]					
NOTES:						
• MAG 72: also called L72						
• MAG 72: Amino acid substitutions that reduce binding 10 fold: 257 T/R or A or G, 262 N/T, 368 D/R or T, 370 E/R or Q, 384 Y/E, 421 K/L, 477 D/V – neutralizes MN, IIB and RF [Kang et al.(1994)]						
• MAG 72: Called L72 – used to bind gp120 to solid phase to select MAbs from a phage selection library [Ditzel et al.(1997)]						
546 MAG 86	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	sCD4-(rHXB2 gp120)-complex	murine(unk)
Donor:	C. Y. Kang, IDEC Inc					
References:	[Kang et al.(1994)]					
NOTES:						
• MAG 86: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 421 K/L, 470 P/L, 477 D/V – neutralizes MN, IIB and RF [Kang et al.(1994)]						

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immuno	Species(Isootype)
547 MAG 96	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	sCD4-(rHXB2 gp120)-complex	murine(unk)
Donor:	C. Y. Kang, IDEC Inc					
References:	[Kang et al.(1994)]					
NOTES:						
• MAG 96: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, 368 D/R or T, 370 E/R – weak neutralization of IIIB [Kang et al.(1994)]						
548 MAG 116	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	sCD4-(rHXB2 gp120)-complex	murine(unk)
Donor:	C. Y. Kang, IDEC Inc					
References:	[Kang et al.(1994)]					
NOTES:						
• MAG 116: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R, 368 D/R or T, 370 E/R – neutralizes MN, IIIB and RF [Kang et al.(1994)]						
549 MAG 3B	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	N	sCD4-(rHXB2 gp120)-complex	murine(unk)
Donor:	C. Y. Kang, IDEC Inc					
References:	[Kang et al.(1994)]					
NOTES:						
• MAG 3B: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R or A or G, 262 N/T, 368 D/R or T, 370 E/R or Q, 381 E/P, 384 Y/E, 421 K/L, 475 M/S, 477 D/V [Kang et al.(1994)]						
550 MAG 12B	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	sCD4-(rHXB2 gp120)-complex	murine(unk)
Donor:	C. Y. Kang, IDEC Inc					
References:	[Kang et al.(1994)]					
NOTES:						
• MAG 12B: Amino acid substitutions that reduce binding 10 fold: 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 477 D/V – weak neutralization of IIIB [Kang et al.(1994)]						

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immuno	Species(Isootype)
551 MAG 29B	gp120(CD4BS dis)	gp120(dis)	DISCONTINUOUS	L	sCD4-(rHXB2 gp120)-complex	murine(unk)
Donor: C. Y. Kang, IDEC Inc References: [Kang et al.(1994)]						
NOTES: • MAG 29B: Amino acid substitutions that reduce binding 10 fold: 257 T/R, 368 D/R or T, 370 E/R or Q, 384 Y/E, 386 N/Q, 421 K/L – weak neutralization of IIIB [Kang et al.(1994)]						
552 120-1B1	gp120(CD4BS dis)		DISCONTINUOUS	L		human(unk)
Donor: Virus Testing Systems Corp., Houston, TX References: [Watkins et al.(1993)]						
NOTES: • 120-1B1: A neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – 120-1B1 was not affected by this mutation [Watkins et al.(1993)]						
553 MAG 6B	gp120(dis)	gp120(dis)	DISCONTINUOUS	N	sCD4-(rHXB2 gp120)-complex	murine(unk)
Donor: C. Y. Kang, IDEC Inc References: [Kang et al.(1994)]						
NOTES: • MAG 6B: Amino acid substitutions that reduce binding 10 fold: 256 S/Y, 257 T/R or G or A, 262 N/T, 368 D/R or T, 370 E/R or Q, 381 E/P, 384 Y/E, 421 K/L, 475 M/S, 477 D/V [Kang et al.(1994)]						
554 P43110	gp120(dis)	gp120(dis)	DISCONTINUOUS	unk	()	()
Donor: Advanced Biosciences (Kensington, MD) References: [di Marzo Veronese et al.(1992), VanCott et al.(1995)]						
NOTES: • P43110: Does not recognize denatured form of the gp120 protein [VanCott et al.(1995)]						

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
555 17b	gp120(CD4i dis)	gp120(dis)	DISCONTINUOUS	L P (weak)	HIV-1 infection	human(unk)

Donor: J. Robinson, Tulane University, LA

References: [Thali et al.(1993), Moore et al.(1993c), Thali et al.(1994), Beretta & Dagleish(1994), Wyatt et al.(1995), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Wu et al.(1996), Trkola et al.(1996a), Binley et al.(1997), Fouts et al.(1997), Li et al.(1997), Weinberg et al.(1997), Ditzel et al.(1997)]

NOTES:

- 17b: 48d and 17b have similar epitopes, and the pair are unique among human and rodent MAbs
- 17b: Epitope is better exposed upon CD4 binding to gp120 – competes with 15e and 21h, anti-CD4 binding site MAbs – 113 D/R, 252 R/W, 257 T/A or G, 370 E/D, 382 F/L, 420 I/R, 433A/L, 438 P/R and 475 M/S confer decreased sensitivity to neutralization [Thali et al.(1993)]
- 17b: Binding of 48d is much more influenced by sequence variation among molecular clones of LAI than is binding of 17b [Moore et al.(1993c)]
- 17b: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs F105, 48d, 21h and 15e) [Thali et al.(1994)]
- 17b: Studies using a V1/V2 deletion mutant demonstrated that enhanced binding of 17b in the presence sCD4 involves the V1/V2 loops, with more significant involvement of V2 – similar effect observed for 48d and A32 [Wyatt et al.(1995)]
- 17b: Binds with higher affinity to monomer and oligomer, slow association rate, poor neutralization of lab strain – this is in contrast to 48d, which has very different kinetics [Sattentau & Moore(1995)]
- 17b: Many MAbs inhibit binding (anti-C1, -C5, -C4, -CD4BS) – anti-V3 MAb 5G11 enhances binding, as do C1-C4 discontinuous epitopes A32 and 2/11c – enhances binding of some anti-V2 MAbs [Moore & Sodroski(1996)]
- 17b: Binding did not result in significant gp120 dissociation from virion, in contrast to 48d, although the gp41 epitope of MAb 50-69 was exposed [Poignard et al.(1996a)]
- 17b: MIP-1 α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4 – binding of 17b blocks this inhibition [Wu et al.(1996)]
- 17b: Neutralizes JR-FL – inhibits gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola et al.(1996a)]
- 17b: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)]
- 17b: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 17b bound monomer, oligomer, and neutralized JRFL in the presence of sCD4, but if sCD4 was not present, 17b only bound monomer [Fouts et al.(1997)]
- 17b: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 17b has synergistic response in combination with anti-V3 MAb 694/98-D [Li et al.(1997)]
- 17b: 48d binds to the IIIB protein and not IIIB V3 peptide, while binding to the Can0A V3 peptide, suggesting Can0A V3 is a conformer that mimics the 48d, but not 17b, epitope [Weinberg et al.(1997)]

HIV Monoclonal Antibodies

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
556 48d	gp120(CD4i dis)	gp120(dis)	DISCONTINUOUS	L P (weak)	HIV-1 infection	human(IgG _{1,κ})

Donor: J. Robinson, Tulane University, LA

References: [Thali et al.(1993), Moore & Ho(1993), Moore et al.(1993c), Thali et al.(1994), Moore et al.(1994b), D'Souza et al.(1995), Sattentau(1995), Wyatt et al.(1995), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard et al.(1996a), Trkola et al.(1996a), Binley et al.(1997), Li et al.(1997), Weinberg et al.(1997), Lee et al.(1997), Ugolini et al.(1997)]

NOTES:

- 48d: Also called 4.8d and 4.8D
- 48d: 48d and 17b have similar epitopes, and the pair are unique among human and rodent MAbs
- 48d: Epitope is better exposed upon CD4 binding to gp120 – competes with ICR 39.13, 15e and 21h, anti-CD4 binding site MAbs – inhibited by anti-CD4BS MAb ICR 39.13g and linear anti-C4 MAbs G3-42 and G3-508 – 113 D/R, 252 R/W, 257 T/A or G, 370 E/D, 382 F/L, 420 I/R, 421 K/L, 433A/L, 438 P/R and 475 M/S confer decreased sensitivity to neutralization [Thali et al.(1993)]
- 48d: Called 4.8d – Neutralizes IIIB – reactive with SF-2 gp120 – does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)]
- 48d: Binding of 48d is much more influenced by sequence variation among molecular clones of LAI than is binding of 17b [Moore et al.(1993c)]
- 48d: A mutation in gp41, 582 A/T, confers resistance to neutralization (also confers resistance to MAbs F105, 21h, 15e and 17b) [Thali et al.(1994)]
- 48d: Poor cross-reactivity with gp120 from most clades [Moore et al.(1994b)]
- 48d: Called 4.8D – Found to neutralize MN, but not JRCSF, two B subtype primary isolates, or a D subtype primary isolate, by most labs in a multi-laboratory study involving 11 labs[D'Souza et al.(1995)]
- 48d: Studies using a V1/V2 deletion mutant demonstrated that enhanced binding of 48d in the presence of sCD4 involves the V1/V2 loops, with more significant involvement of V2 – similar effect observed for 17b and A32 [Wyatt et al.(1995)]

MAb ID	556 48d (cont.)
	<ul style="list-style-type: none"> • 48d: Formalin inactivation of virus at 0.1% formalin for 10 hours at 4 degrees was optimal for inactivation of virus while maintaining epitope integrity [Sattentau et al.(1995)] • 48d: Binds with similar affinity to monomer and oligomer, moderate association rate, potent neutralization – this is in contrast to 17b, which has very different kinetics [Sattentau & Moore(1995)] • 48d: Many MAbs inhibit binding (anti-C1, -C5, -C4, -CD4BS) – anti-C1-C4 discontinuous epitope MAbs A32 and 2/11c enhance binding – reciprocal enhanced binding with some anti-V2 MAbs [Moore & Sodroski(1996)] • 48d: Binding resulted in gp120 dissociation from virion, mimicking sCD4, and exposure of the gp41 epitope of MAb 50-69, in contrast to CD4BS MAbs [Poignard et al.(1996a)] • 48d: Neutralizes JR-FL – slightly inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola et al.(1996a)] • 48d: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)] • 48d: One of 14 human MAbs tested for ability to neutralize a chimeric HIV-vpu+, which expressed HIV-1 IIIB env – all Ab combinations tested showed synergistic neutralization – 48d has synergistic response with MAbs 694/98-D (anti-V3) and F105 [Li et al.(1997)] • 48d: 48d binds to the IIIB protein and not IIIB V3 peptide, while binding to the Can0A V3 peptide, suggesting Can0A V3 is a conformer that mimics the 48d, (but not 17b), epitope [Weinberg et al.(1997)] • 48d: Prefers CD4-gp120 complex to gp120 alone, but does not enhance fusion, in contrast to MAb CG10, in fact it inhibits syncytium formation [Lee et al.(1997)] • 48d: Viral binding inhibition by 48d was strongly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini et al.(1997)] • 48d: NIH AIDS Research and Reference Reagent Program: 1756

HIV Monoclonal Antibodies

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
557 A32	gp120(CD4i C1-C4 dis)	gp120(dis)	DISCONTINUOUS	N	?	human(unk)
Donor: J. Robinson, Tulane University, LA						
References: [Moore et al.(1994b), Wyatt et al.(1995), Moore & Ho(1995), Moore & Sodroski(1996), Wu et al.(1996), Trkola et al.(1996a), Binley et al.(1997), Fouts et al.(1997)]						
NOTES:						
• A32: Reacted with virtually every gp120 monomer of every clade tested, most conserved gp120 monomer epitope known [Moore et al.(1994b)]						
• A32: Epitope is better exposed upon CD4 binding to gp120 – binding of A32 enhances binding of 48d and 17b – studies using a V1/V2 deletion mutant demonstrated that enhanced binding of 48d in the presence sCD4 involves the V1/V2 loops, with more significant involvement of V2 [Wyatt et al.(1995)]						
• A32: Review: epitope is distinct from CD4BS MAbs, 48d and 17b, and 2G12 [Moore & Ho(1995)]						
• A32: Reciprocal inhibition of binding of anti-C1, -C5, -V3 and anti-CD4 binding site MAbs – induces binding of some anti-V2 and sCD4 inducible MAbs (48d and 17b) – very similar competition pattern to 2/11c, A32 and 211/c are unique among known human and rodent MAbs [Moore & Sodroski(1996)]						
• A32: Not neutralizing – binds domains that interact with gp41 – MIP-1 α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4 and binding of A32 does not block this inhibition [Wu et al.(1996)]						
• A32: Does not neutralize JR-FL, or any strain strongly – partial inhibition of gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola et al.(1996a)]						
• A32: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)]						
• A32: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric env binding – A32 bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)]						
558 2/11c	gp120(C1-C4 dis)	gp120(dis)	DISCONTINUOUS	L (weak)	HIV-1 infection	human(unk)
Donor: J. Robinson, Tulane University, LA						
References: [Moore & Sodroski(1996), Trkola et al.(1996a), Binley et al.(1997), Fouts et al.(1997), Li et al.(1997)]						
NOTES:						
• 2/11c: 2/11c is also called 211c, 2.11c and 2-11c						
• 2/11c: Inhibits binding of anti-C1, -C5, -C4, -V3 and anti-CD4 binding site MAbs – induces binding of some anti-V2 and CD4i MAbs (48d and 17b) – similar reactivity pattern to A32, but less cross-reactive and lower affinity – A32 and 211/c are unique among known human and rodent MAbs [Moore & Sodroski(1996)]						
• 2/11c: Called 211c – does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1 β -CCR-5 competition study [Trkola et al.(1996a)]						
• 2/11c: A low avidity antibody as assessed by urea elution; study indicated that MAbs with discontinuous binding sites tended to have low avidity [Binley et al.(1997)]						
• 2/11c: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric env binding – 2/11c bound monomer, did not bind oligomer or neutralize JRFL [Fouts et al.(1997)]						
• 2/11c: Called 2.11c – One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 50% neutralization could not be achieved at a maximal concentration of 67 μ g/ml [Li et al.(1997)]						

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunocon-	Species(Isootype)
559 N70-2.3a	gp120(272-509 dis)	gp120(dis)	DISCONTINUOUS	N	HIV-1 infection	human(IgG1)
Donor:	J. Robinson, Tulane University, LA					
References:	[Robinson et al.(1990), Takeda et al.(1992)]					
NOTES:						
• N70-2.3a: Broad reactivity [Robinson et al.(1990)]						
• N70-2.3a: Fc receptor mediated enhancement of HIV-1 infection – binds a conformational site in the carboxyl half of gp120, distinct from 1.5e [Takeda et al.(1992)]						
560 6E10	gp120 (dis)	gp120(dis)	DISCONTINUOUS	L	rsgp160	(unk)
Donor:	Phil Berman					
References:	[Berman et al.(1991)]					
NOTES:						
• C31: Broadly reactive group specific – high yield cultivation of human MAb [Boyer et al.(1991)]						
561 C31	gp120(unknown)	gp120	?	N	HIV-1 infection	human(IgG _{1κ})
Donor:	Evan Hersh					
References:	[Boyer et al.(1991)]					
NOTES:						
• C31: Broadly reactive group specific – high yield cultivation of human MAb [Boyer et al.(1991)]						
562 P5-3	gp120(unknown)	gp120	?		HIV-1 infection	human(IgG _λ)
Donor:	Evan Hersh and Yoh-Ichi Matsumoto					
References:	[Robinson Jr. et al.(1990a), Pincus et al.(1991)]					
NOTES:						
• P5-3: No enhancing activity for HIV-1 IIIB [Robinson Jr. et al.(1990a)]						
• P5-3: Poor immunotoxin activity when coupled to RAC – isotype specified as: IgG _{3λ} [Pincus et al.(1991)]						
• P5-3: NIH AIDS Research and Reference Reagent Program: 378						
563 BAT401	gp120(unknown)	gp120	?	L	Intact IIIB	murine(IgG1)
Donor:	Fung et al.(1987)					
References:	[Fung et al.(1987)]					
NOTES:						
• BAT401: No enhancing activity for HIV-1 IIIB [Fung et al.(1987)]						
564 BAT267	gp120(unknown)	gp120	?	L	Inact IIIB	murine(IgG1)
Donor:	Fung et al.(1987)					
References:	[Fung et al.(1987)]					
NOTES:						
• BAT267: No enhancing activity for HIV-1 IIIB [Fung et al.(1987)]						
565 BAT509	gp120(unknown)	gp120	?	L	Inact IIIB	murine(IgG1)
Donor:	Fung et al.(1987)					
References:	[Fung et al.(1987)]					
NOTES:						
• BAT509: No enhancing activity for HIV-1 IIIB [Fung et al.(1987)]						

HIV Monoclonal Antibodies

MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
566 13.10	gp120(unknown) Donor: Evan Hersh and Yoh-Ichi Matsumoto References: [Lake et al.(1989), Moran et al.(1993), Wisniewski et al.(1996)]	gp120 ?	N	HIV-1 infection	human(IgG ₁ _λ)	
	NOTES:					
	• 13.10: Also called No. 13					
	• 13.10: First HIV-1 specific human-mouse hybridoma that produces a MAb that binds to gp120 and gp160 [Lake et al.(1989)]					
	• 13.10: Heavy (V _H I) and light (V _λ II) chain sequenced – no enhancing or neutralizing activity – called No. 13					
	• 13.10: Heavy (V _H I) and light (V _λ II) chain sequenced [Moran et al.(1993)]					
	• 13.10: 13.10 is V _H 1 – V-region heavy chain usage was examined and a bias of enhanced V _H 1 and V _H 4, and reduced V _H 3, was noted among HIV infected individuals [Wisniewski et al.(1996)]					
	• 13.10: NIH AIDS Research and Reference Reagent Program: 377					
567 F285	ENV(unknown) References: [Wisniewski et al.(1995), Wisniewski et al.(1996)]	gp120 ?		HIV-1 infection	human(IgG ₁)	
	NOTES:					
	• F285 is V _H 1 – V-region heavy chain usage was examined and a bias of enhanced V _H 1 and V _H 4, and reduced V _H 3, was noted among HIV infected individuals [Wisniewski et al.(1996)]					
568 HBW4	gp120(unknown IIIB) References: [Moran et al.(1993), Wisniewski et al.(1995), Wisniewski et al.(1996)]	gp120 ?		HIV-1 infection	human(IgG ₁ _λ)	
	NOTES:					
	• HBW4: Heavy (V _H II) and light (V _λ II) chain sequenced [Moran et al.(1993)]					
	• HBW4: HBW4 is V _H 2 – V-region heavy chain usage was examined and a bias of enhanced V _H 1 and V _H 4, and reduced V _H 3, was noted among HIV infected individuals [Wisniewski et al.(1996)]					
569 multiple Fab's	gp120(unknown) References: [Burton et al.(1991)]	gp120 ?		HIV-1 infection	human(unk)	
	NOTES:					
	• multiple Fab's: A panel of anti-gp120 Fab's was generated by antigen selection from a random combinatorial library prepared from bone marrow from an asymptomatic individual [Burton et al.(1991)]					

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MAb ID	Location	WEAU	Sequence	Neutralizing	Immunogen	Species(Isotype)
570	multiple MAbs	gp120(unknown)	gp120 ?		gp120 complexed with MAb M77	murine(unk)
	References: [Denisova et al.(1996)]					
	NOTES:					
	• multiple MAbs: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes, as well as an array of MAbs to discontinuous epitope – 10 of 36 MAbs were mapped to linear epitopes and are mentioned elsewhere in this database, the others are: GV5H1, GV4D5, GV4G10, GV1A8, GV10H5, GV8E11, GV2H4, GV6E6, GV1F7, GV1G9, GV4G5, GV6B12, GV1E8, GV2B7, GV1B11, GV6H5, GV6G2, GV6B5, GV1E10, GV5E3, GV5B9, GV5F4, GV6G4, GV1A12, GV5C11, GV6B6, GV3C10 [Denisova et al.(1996)]					
571	multiple MAbs	gp120(dis)	gp120 ?	gp120		murine(unk)
	References: [Denisova et al.(1996)]					
	NOTES:					
	• multiple MAbs: When gp120 was used as an immunogen, in contrast to gp120 bound to an anti-V3 MAb, few MAbs were generated and all bound better to the native than to the denatured protein – MAbs generated were: G1B12, G2F7, G9G8, G12F12, G1B8, G11F11, G9E8, G1B11, G1B6, G6F2, G2E7 [Denisova et al.(1996)]					
572	multiple MAbs	gp120(dis)	gp120(dis) ?		gp120-CD4 complex	murine(unk)
	References: [Denisova et al.(1996)]					
	NOTES:					
	• multiple MAbs: When gp120-CD4 was used as an immunogen, in contrast to gp120 bound to an anti-V3 MAb, few MAbs were generated and all bound better to the native than to the denatured protein – MAbs generated were: CG43, CG41, CG49, CG53, CG42, CG4, CG46, CG40, CG51, CG48, CG50, CG125, CG124, CG121 [Denisova et al.(1996)]					